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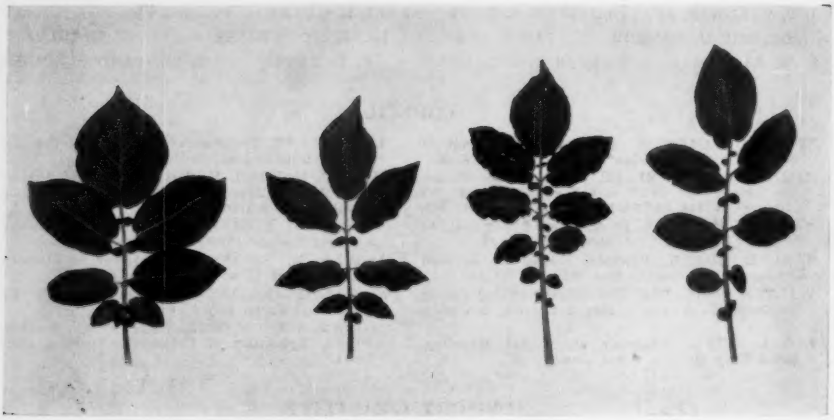

HAPLOID POTATO LEAVES

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Loose Pericarp in Corn

Linkage in the Honey Bee

Scurred Aberdeen-Angus Cattle

Albino-Dwarfism in Hereford Cattle

The Origin of the B Genome of Wheat

Effect of Temperature on Mutation Rate in Corn

VOLUME 49 MAY-JUNE, 1958 NUMBER 3

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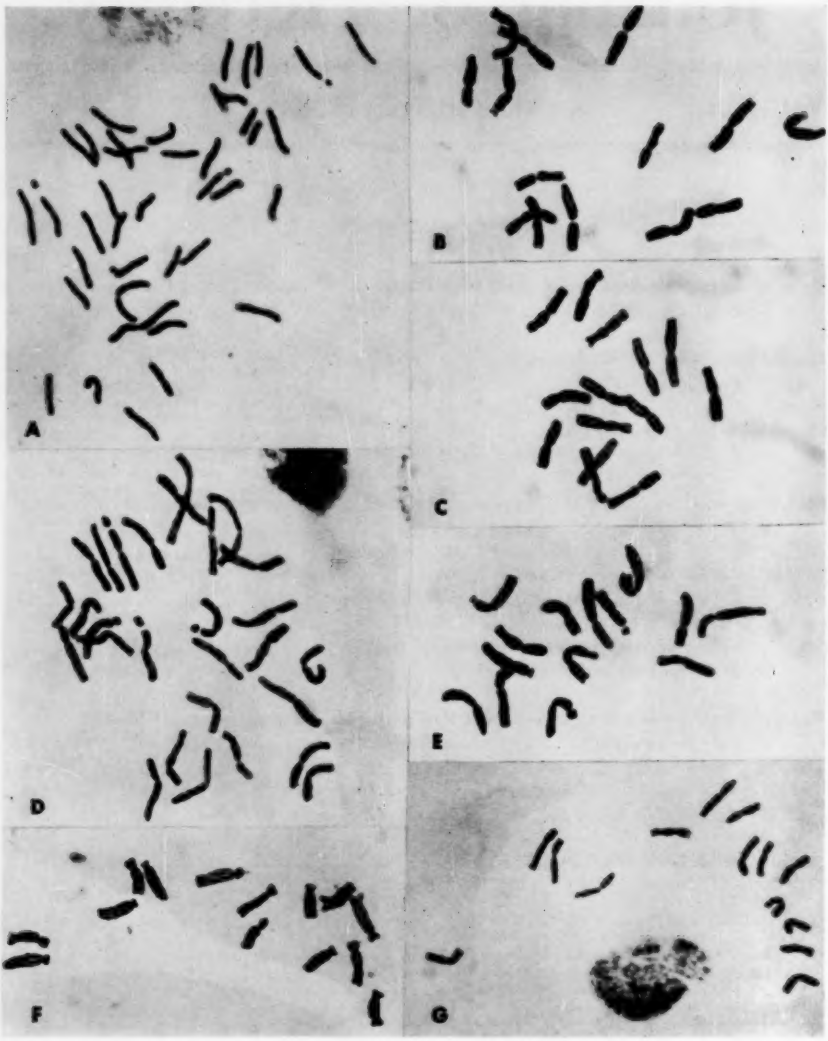

MITOSES FROM ROOT TIPS

Figure 1

A-G—Shows metaphases of mitosis in root-tips pretreated with bromo-naphthalene. *A—Triticum vulgare; B—T. aegilopoides; C—Aeg. speltoides; D—T. turgidum; E—Aeg. bicornis; F—Agropyron triticeum; G—Aeg. squarrosa.* (*A* × 750; *B-G* × 1200).

EVIDENCE ON THE ORIGIN OF THE B GENOME OF WHEAT

RALPH RILEY, JOHN UNRAU AND VICTOR CHAPMAN*

THE sub-tribe Triticinae of the Graminae includes the genera *Triticum, Aegilops, Agropyron, Secale* and *Haynaldia* which incorporate certain agriculturally important species. Four of these genera contain polyploid series of species and the basic chromosome number is seven throughout. The wheat genus, *Triticum*, has diploid, tetraploid and hexaploid species with 14, 28 and 42 chromosomes, constituted of the *AA*, *AABB* and the *AABBDD* genomes respectively.

The close relationships of the *A* genome in tetraploid, hexaploid and diploid wheat is well established, and *Aegilops squarrosa* has been shown by the independent studies of McFadden and Sears[17] and Kihara[10] to be the source of the *D* genome in hexaploid wheats. A number of investigators have speculated on the source of the *B* genomes of the tetraploid and hexaploid wheats. On the basis of chromosome pairing at meiosis in F_1 hybrids involving tetraploid and hexaploid wheats with polyploid species of *Agropyron*, Peto[22], Wakar[40] and Matsumura[16] believed that the *B* genome must have been contributed by *Agropyron*. McFadden and Sears[18] suggested that the species involved might be the diploid *Agr. triticeum*. Using Anderson's method of extrapolation[1] Sarkar and Stebbins[26] concluded that the species morphologically closest to the extrapolate was *Aegilops speltoides*.

Recently, Sears[31] has suggested that *Aegilops bicornis* was the source of the *B* genome because of the close morphological resemblance of the amphiploid of *T. monococcum* × *Aeg. bicornis* to *T. dicoccum*.

In this paper, evidence will be presented on the karyotypes and the behavior in hybridization of a number of possible *B* genome contributing species.

Materials

The species which have been directly involved in the present work are listed below together with their chromosome numbers and the genome symbols assigned to them by Kihara[15]. The names in parentheses are those used in the text to avoid confusion between different forms of the same species.

Species	Chromosome number (n)	Genome symbol
Triticum monococcum L. var. *macedonicum* Papeg.	7	*A*
T. boeticum (Boiss.) Schiem. sub. sp. *aegilopoides* (Link) Schiem. (= *T. aegilopoides*)	7	*A*
T. boeticum (Boiss.) Schiem. sub. sp. *thaoudar* (Reuter) Schiem. (= *T. thaoudar*)	7	*A*
T. dicoccoides Körn. var. *kotschyanum* Perc.	14	*AB*
T. dicoccum Schubl. *farrum* Körn.	14	*AB*
T. dicoccum Schubl. sub. sp. *georgicum* Dekap. et Men. (= *T. georgicum*)	14	*AB*
T. durum Desf. var. *caerulescens* Körn.	14	*AB*
T. turgidum L. var. *iodurum* Körn.	14	*AB*
T. timopheevi Zhuk. var. *typicum* Zhuk.	14	*AG*
Aegilops speltoides Tausch. var. *aucheri* (Boiss.) Born.	7	*S*
Aeg. speltoides Tausch. var. *ligustica* (Savegna) Coss.	7	*S*
Aeg. longissima Schw. et Muschl.	7	S^l
Aeg. bicornis (Forsk.) Jaub. et Sp.	7	S^b
Aeg. sharonensis Eig.	7	S^b
Agropyron triticeum J. Graertn.	7	?

Karyotypes

The different genomes of wheat and most of its close relatives are composed of chromosomes, with median or submedian centromeres, which lack distinctive features of morphology. While there are differences in total length and to some extent in arm length ratios, except

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for the satellited chromosomes, there are no readily observed diagnostic features in the chromosome complement of wheat. Few detailed karyotype studies have, consequently, been undertaken on wheat and its relatives. In tetraploid and hexaploid species, three pairs of satellited chromosomes were observed by Bhatia[3] Pathak[21] and Camara[4], while Livitzky *et al.*[14] and Tjio and Levan[38], and most recent authors, have noted only two pairs.

Morrison[20] has identified the two satellited pairs most commonly observed as I and X of Chinese Spring. Quite recently Okamoto[42] has confirmed Larson's hypothesis[13] that these chromosomes both belong to the *B* genome.

If the morphology of the satellited chromosomes has undergone little or no change since their incorporation in the *B* genome of tetraploid wheats, then the donor of *B* must be a diploid species with two pairs of satellited chromosomes similar in morphology to those of polyploid wheat.

The karyotypes have been studied in a number of diploid species that have been considered as possible donors of the *B* genome in wheat. Figure 1 *A-G* shows photographs of mitoses from root-tips, which were pretreated three to four hours in saturated solutions of bromonapthalene, prior to fixation in glacial acetic acid, and hydrolysed in normal HCl at 60°C for 12 minutes prior to staining in basic fuchsin.

The satellited pair of chromosomes in *T. aegilopoides* is quite different from either of those of 4.r and 6.r wheat in the size of the satellite and in arm length ratio, indicating definitely that the *A* genome could not have become the *B* genome through doubling and subsequent modification of one genome. Since the satellited chromosomes of tetraploid and hexaploid wheats are similar, and the satellited pairs are in the *B* genome, the *D* genome of *Aeg. squarrosa* as a source of one pair even in the hexaploids species can be ruled out. Moreover the satellites of *Aeg. squarrosa* are much smaller than those of polyploid wheat and are apparently not seen in hexaploid wheats.

As indicated earlier, *Agropyron triticeum* has been suggested as the diploid with the *B* set. On the basis of karyotype, however, this is untenable as Sarkar and Stebbins[26] have already pointed out. Wheat has no chromosomes with subterminal centromeres like those of this species (Figure 1*F*) which consequently need not be regarded as being closely related to wheat.

Characters associated with the tetraploid but not with the diploid wheats are found only in the Sitopsis section of *Aegilops*, in combination with a karyotype composed entirely of chromosomes with median or submedian centromeres. It is in this section, therefore, that detailed karyotype studies have been made.

The members of the Sitopsis section of *Aegilops*, on the basis of karyotype and plant morphology, are of two main types with a further subdivision of one of the main groups.

1. *Aeg. speltoides* var. *ligustica* has two pairs of chromosomes with fairly large satellites. This form is fully awned and the rachis is brittle below each node. Variety *aucheri* has a similar karyotype but only the apical spikelet is awned, and the rachis is brittle only below the basal nodes.
2. (a). *Aeg. sharonensis* and *Aeg. longissima* have similar karyotypes which include one pair of chromosomes with large and one pair with distinctly smaller satellites. In awning and rachis brittleness *Aeg. sharonensis* and *Aeg. longissima* parellel *Aeg. speltoides* var. *ligustica* and var. *aucheri* respectively.

   (b). *Aeg. bicornis* is similar to *Aeg. sharonensis* and *Aeg. longissima* in karyotype, but is apparently a morphologically monotypic species.

The morphology of the satellited chromosomes of *Aeg. bicornis*, *Aeg. sharonensis* and *Aeg. longissima* does not correspond to that of the satellited chromosomes of the *B* genome since all of these species have a similar pair with very small satellites.

Aegilops speltoides, on the other hand,

fits the requirement of the donor of the satellited chromosomes perfectly, as can be seen in Figure 1 in which the chromosomes of this species as well as those of tetraploid and hexaploid wheats are shown. The karyotypes of *Aeg. speltoides* and the *B* genome are similar in that the longer pair of satellited chromosomes, chromosome I of *T. vulgare*, has the smaller satellites and is the more heterobrachial. By contrast the other satellited pair, chromosome X of *T. vulgare*, is shorter but with longer satellites. The satellites of I are less than one-third the length of the adjacent arm, whereas in X the satellites are one-third to one-half the length of the adjacent arm. Clearly, if *Aeg. speltoides* is the source of *B*, little or no change in these chromosomes has occurred since its incorporation to form the tetraploids. Moreover, in preparations of the amphiploid *T. monococcum* × *Aeg. speltoides*, chromosome morphology, especially of the satellited chromosomes, is strikingly similar to that of emmer. Consequently, on the basis of karyotype, *Aegilops speltoides* or one of its relatives is the species most closely approaching the requirements of the donor of the *B* genome of polyploid wheat.

Chromosome Pairing in Hybrids

The genome concept as originally propounded by Kihara[8] for wheat and its relatives requires that the haploid sets of chromosomes of cytologically distinct diploid species retain their separate identities when combined to form allopolyploids. Similarity would be expressed as a high incidence of meiotic pairing of chromosomes of the diploid set with their homologues of the corresponding genome of the polyploid, in hybrids between the polyploid and a constituent diploid ancestor. Distinctness or similarity of genomes is not, however, always expressed with unambiguous exactness, for grades of cytological relationships may exist from complete homology to absolute distinctiveness. Genome formulae, therefore, should not be interpreted as indicating absolute identity or difference, but the formulae, nevertheless, permit a general appreciation of phylogenetic and experimental situations. The highly successful genome analysis of *Aegilops* by Kihara[11] and his collaborators has had two aspects; first the recognition of cytologically distinct diploid species, and secondly, the determination of how diploids have associated together to form the allopolyploid species. The artificial synthesis of several polyploid species, for example *Aeg. cylindrica*[30], from their putative parents has confirmed the results of genome analysis. The genome analysis of tetraploid and hexaploid wheat, however, has thus far been only partially successful.

It was apparent to the early investigators, Sax[27] and Kihara[7], that the haploid complement of the tetraploid wheats consisted of the seven chromosomes of diploid wheat, the *A* genome, plus a further set of seven, the *B* genome. Also the haploid complement of the hexaploid wheats comprised the 14 chromosomes present in the tetraploids, the *AB* genomes, plus a further seven chromosomes, the *D* genome. It may be pointed out here that all diploid wheats are genomically similar, forming fully fertile hybrids with normal meiosis. Most tetraploid wheat species also form fertile hybrids with normal meiotic pairing. There are, however, two species, *T. timopheevi* and *T. armeniacum* which are genomically somewhat distinct and are described as having the *AG* rather than the *AB* genomes, but in the present context, "tetraploid wheat" will imply *AB* species. All hexaploid wheat species have the genome formula *ABD*. McFadden and Sears[17] and Kihara and Lilienfeld[12] produced synthetic *T. spelta* by doubling the chromosome number of the F_1 from crosses of tetraploid wheat with *Aeg. squarrosa*. This demonstrated the efficacy of the genome analyses which had lead them to suppose that *Aeg. squarrosa* had contributed the *D genome*[10]. Genome analysis, based on meiotic pairing in hybrids has however, so far, failed to reveal the source of the *B* genome.

While meiotic pairing between the chromosomes of diploid wheats and those of the *A* genome of the tetraploids is not complete (Table I) it is nevertheless sufficient to indicate that the tetraploids derive this genome from the diploids[26]. Undoubtedly, some cytological and genetical divergence of the *A* genomes, of diploids and of tetraploids, has occurred since the formation of the tetraploids and this may account for the incomplete pair-

TABLE I. Chromosome pairing in wheat triploid hybrids

Hybrid	Cells	Mean univalents	Mean bivalents	Mean trivalents
T. georgicum × *T. thaoudar*	30	9.77 ± 0.76	5.47 ± 0.15	0.10
T. georgicum × *T. aegilopoides*	30	10.63 ± 0.36	4.93 ± 0.19	0.17
T. georgicum × *T. monococcum*	30	11.37 ± 0.36	4.67 ± 0.18	0.10

ing of *A* genome chromosomes in hybrids of 4*x* × 2*x* wheats. It is, consequently, reasonable to assume that no greater diversity will have developed between the chromosomes of the *B* genome of tetraploid wheat and those of the contemporary diploid descendants of the original *B* genome contributor. It is to be expected, therefore, that triploid hybrids between tetraploid wheats and contemporary descendants of the *B* genome-contributing diploid should show meiotic pairing similar to that observed in hybrids between diploid and tetraploid wheats. The occurrence of such pairing in a hybrid would indicate the source of the *B* genome.

Apparently the hybrid between tetraploid wheat and the diploid species *Agropyron triticeum* has never been made, although many efforts have been expended in attempting the cross since McFadden and Sears[18] suggested this species as the source of *B*. Clearly the failure of the cross indicates a considerable degree of distinctness especially since there is wide cross-compatibility in the Triticinae.

In a hybrid between the diploid species *Agr. elongatum* and a tetraploid wheat, Jenkins and Mochizuki[6] have reported a mean of 2.6 bivalents per cell at meiosis. Such pairing may well be accounted for by autosyndesis of parental chromosomes such as is indicated by pairing in haploids of tetraploid wheat[7, 24]. There can be little homology between the chromosomes of *Aeg. elongatum* and those of the tetraploid; indeed, Jenkins and Mochizuki[6] suggested a separate symbol for the genome of this species to indicate its remoteness. Further evidence against *Agropyron* as the source of *B* is the fact that apparently no character transfer from *Agropyron* to wheat has occurred in the many breeding investigations involving hybrids of tetraploid and hexaploid wheats with polyploid *Agropyrons*. Stebbins and Pun[33] have demonstrated that the pairing observed in such hybrids probably resulted entirely from the autosyndesis within the *Agropyron* set. The sum of the evidence, therefore, indicates that there is no genome in *Agropyron* closely related to a wheat genome.

Evidence from karyotypes indicates a member of the Sitopsis section as the likely contributor of the *B* genome, and data on chromosome pairing in hybrids of tetraploid wheats with members of the Sitopsis section are summarized in Table II.

As indicated earlier the Sitopsis section of the genus *Aegilops* consists of three rather well defined groups. On the basis of chromosome pairing and fertility in hybrids within the section they again fall into the same groups with the following distinctive behavior patterns.

1. *Aeg. speltoides* var. *ligustica* and var. *aucheri* form fully fertile hybrids with normal meiotic behavior.

2. (a). *Aeg. longissima* and *Aeg. sharonensis* differ by one translocation but otherwise their hybrids have normal meiotic pairing and fertility. The two species will be referred to as *longissima—sharonensis*.

(b). Good pairing at meiosis in hybrids indicates that *Aeg. bicornis* is quite close to *longissima—sharonensis* but some genetic divergence is revealed by low hybrid-fertility.

In general, pairing in hybrids from intercrossing all members of the Sitopsis section has been found to be high[24, 34]; and consequently Kihara[11] grouped them all together with the same primary genome symbol, *S*. The species *Aeg. sharonensis*, *Aeg. longissima* and *Aeg. bicornis* form fertile hybrids with one another, whereas hybrids of any of these species with *Aeg. speltoides* are infertile. Thus it is clear that *Aeg. sharonensis—longissima—bicornis* are of close affinity, while *Aeg. speltoides* is more remote.

In hybrids of *Aeg. bicornis* with tetraploid wheat only 1.2 bivalents per cell are found (Table II) indicating little or no homology between the chromosomes of *Aeg. bicornis* and those of tetraploid wheat. Similarly, hybrids of *longissima—sharonensis* with tetraploid wheat have very little pairing (Table II). Sears[31] found that hybrids of the amphiploid of einkorn × *Aeg. bicornis* with tetraploid wheat had a mean of only 8.6 bivalents per cell, and Tanaka[34] found five to nine (mean seven) bivalents in hybrids of the amphiploid *Aeg. longissima* × *T. aegilopoides* with *T. durum*. Probably, most of the pairing in these

TABLE II. Chromosome pairing in wheat-*Aegilops* hybrids

Hybrid	Cells	Mean univalents	Mean bivalents	Mean trivalents	Mean quadrivalents
Aeg. speltoides × *T. monococcum*	75	7.25 ± 0.35	3.37 ± 0.17	.....	.....
Aeg. speltoides × *T. dicoccoides*	35	5.94 ± 0.36	5.21 ± 0.26	1.59	0.06
Aeg. speltoides × *T. dicoccum*	50	6.70 ± 0.28	6.22 ± 0.18	0.62	.....
Aeg. speltoides × *T. georgicum*	30	8.00 ± 0.53	4.96 ± 0.28	1.00	0.08
Aeg. speltoides × *T. durum*	50	5.90 ± 0.25	5.96 ± 0.20	1.06	.....
Aeg. speltoides × *T. turgidum*	50	7.92 ± 0.36	5.94 ± 0.18	0.40	.....
Aeg. speltoides × *T. timopheevi*	30	7.60 ± 0.41	6.28 ± 0.19	0.28	.....
T. dicoccoides × *Aeg. bicornis*	40	17.37 ± 0.36	1.78 ± 0.18	0.02	.....
T. turgidum × *Aeg. sharonensis*	40	16.48 ± 0.31	2.22 ± 0.15	0.02	.....
Aeg. longissima × *T. dicoccum*	40	17.43 ± 0.47	1.60 ± 0.17	0.01	.....

hybrids of amphiploids × tetraploid species is accounted for by pairing of the chromosomes of the einkorn genome of the amphiploids with their homologues in the *A* genome of the natural tetraploids.

It is clear from these data that pairing of the order observed between the chromosomes of einkorn and those of the *A* set of the tetraploids does not occur between the chromosomes of the *sharonensis—longissima* and *bicornis* genomes and those of the *B* genome of the tetraploids. On the basis of pairing at meiosis, therefore, it seems unlikely that either the S^1 set of *sharonensis—longissima* or the S^b set of *bicornis* can be considered as the source of the *B* genome.

By contrast, triploid hybrids involving *Aeg. speltoides* with the tetraploids are strikingly different at meiosis from those involving the other Sitopsis species. The results in Table II confirm those obtained by McFadden and Sears[19] and Thompson *et al.*[37] in showing that the level of pairing is fairly high. Thus, an average of only six univalents per cell are present at meiosis in these hybrids in contrast to 16-18 per cell in those involving *sharonensis—longissima* and *bicornis*.

It is evident, however, that the *Aeg. speltoides* genome is partially homologous with both the *A* and *B* genomes of the tetraploids since there is considerable trivalent formation. The degree of homology between the chromosomes of *speltoides* and those of the *A* is indicated by the pairing in hybrids between *Aeg. speltoides* and diploid wheat, (Table II)[29]. But homology with the *B* genome must be as strong, for in hybrids with the tetraploids there are only about the same number of univalents per cell as in hybrids with the diploids.

The closeness of the homologies in these hybrids is expressed also in the hexaploid amphiploids derived from the combinations *Aeg. speltoides* × tetraploid wheats, in which multivalent associations are frequent (Table III)[19]. The genetic instability consequent on multivalent formation was so high that by the third amphiploid generation in the combination *Aeg. speltoides* × *T. durum* not a single plant of the original amphiploid phenotype could be recovered. By contrast, the amphiploids *Aeg. bicornis* × *T. dicoccoides* and *T. turgidum* × *Aeg. sharonensis*, studied at the same time, regularly formed mainly bivalents at meiosis and have been relatively stable phenotypically through seven amphiploid generations. Thus the chromosomal affinities as expressed by pairing and breeding behavior must be much closer in the hybrids with *Aeg. speltoides* than with the other members of the Sitopsis section, suggesting a rather close relationship between the chromosomes of *Aeg. speltoides* and those of the *B* genome.

But if *Aeg. speltoides* is closely related to *B*, why then is there also a fairly high level of pairing with the *A* genome since it is evident that there is little effective homology between the two genomes of the tetraploid wheats, from the pairing in haploids[9, 24]? Little intergenome homology can also be detected from the pairing in certain hybrids, for example those of tetraploid wheat with *Aeg. candata*[2], or with *Agr. elongatum*, Jenkins and Mochizuko[6]. The answer to this and other apparent anomalies has probably been revealed in studies by Riley[23] on certain 20 chromosome nullihaploids and by the work of Sears and Okamoto[32] on crosses between einkorn and a nullisomic of common wheat. These studies show that in the absence of a specific chromosome, which Sears and Okamoto[32] have identified as chromosome V, pairing in the critical haploid and hybrid is very much greater than when this chromosome is present. Very likely this extra pairing is between homoeologous chromosomes of the three different genomes combined in hexaploid wheat. Apparently there is a gene system on chromosome V of common wheat which restricts pairing to chromosomes that are completely homologous, so

TABLE III. Chromosome pairing in wheat-*Aegilops* amphiploids

Amphiploid	Cells	Mean univalents	Mean bivalents	Mean trivalents	Mean quadrivalents	Others
Aeg. speltoides × *T. monococcum*	30	4.03 ± 0.48	10.53 ± 0.22	0.50	0.33	.....
Aeg. speltoides × *T. dicoccoides*	30	3.20 ± 0.38	16.00 ± 0.51	0.73	0.87	0.17V
Aeg. speltoides × *T. dicoccum*	30	4.69 ± 0.61	17.07 ± 0.45	0.58	0.24	0.03V 0.03VI
Aeg. speltoides × *T. durum*	75	7.55 ± 0.53	14.68 ± 0.33	0.78	0.43	0.03V 0.04VI
Aeg. longissima × *T. dicoccum*	30	0.20 ± 0.11	20.90 ± 0.06	.....	.....	.....
T. turgidum × *Aeg. sharonensis*	30	1.00 ± 0.31	20.10 ± 0.23	0.13	0.10	.....

causing hexaploid wheat to behave cytologically like a diploid.

Chromosome V has been shown by Okamoto[42] to belong to the *B* genome, thus the genetic system restricting pairing must also be present in tetraploids. This is supported by the fact that in the many derivatives obtained from pentaploid hybrids there have been no segregants with unusual pairing behavior.

The presence of a genetic system which restricts pairing to closely similar chromosomes makes comprehensible several pairing anomalies in hybrids. Thus, apparently there is homology between some of the chromosomes of *Aeg. squarrosa* and those of the *A* genome, since in hybrids of diploid wheat with *Aeg. squarrosa* means of from 3.2 to 4.9 bivalents per cell were observed by Sears[29]. In contrast, hybrids of *Aeg. squarrosa* with *T. dicoccoides* had an average of only 0.05 bivalents, McFadden and Sears[18], or a maximum of three bivalents per cell[12]. Similarly, in hybrids of *Aeg. ovata* with diploid wheats 3.3 bivalents per cell are formed, whereas in hybrids with a range of tetraploid wheats means of from 0.2 to 0.4 bivalents per cell were formed[2]. Reduced pairing in hybrids involving the tetraploid wheats may well result from genetic restriction of pairing.

A similar situation is found in hybrids involving Sitopsis species. For example in hybrids of *Aeg. bicornis* × diploid wheats there are 2.2 to 2.5 bivalents per cell[19, 25], whereas in hybrids with the tetraploids there are 1.8 bivalents per cell, (see Table II). Similarly in the hybrid *Aeg. sharonensis* × *T. monococcum* Tanaka[35] reports from one to four bivalents per cell, whereas *longissima—sharonensis* × tetraploid wheat hybrids have means of 1.6 to 2.2 bivalents per cell (see Table II). Thus there is a reduction of the potential pairing, even with *A* genome chromosomes, in the presence of the *B* genome. On the other hand, the high pairing of the *A* genome of diploids with the *A* of tetraploids even in the presence of the restrictive system on chromosome V indicates that where close homology exists synapsis can occur.

Aeg. speltoides can be fitted into this scheme if the pairing of its chromosomes with those of the *B* genome is similar to that between chromosomes of the *A* genomes of diploid and tetraploid wheat. It is impossible to demonstrate this clearly because *Aeg. speltoides* chromosomes are apparently not prevented from pairing with chromosomes of the *A* genome in triploid hybrids as is the case in hybrids with other members of the Sitopsis section. Presumably *Aeg. speltoides* contains an allele, or alleles, dominant to those restricting pairing carried on chromosome V. In the triploid hybrids this results in the break-down of the meiotic isolation of the chromosomes of the *A* and the *B* genomes, so that mutual tripartite affinities between homoeologous chromosomes of all three genomes are expressed.

Considerable multivalent formation occurs at meiosis in the amphiploid *T. monococcum* × *Aeg. speltoides* (Table III) because of pairing between chromosomes of the *S* and the *A* genomes. However, this amphiploid is relatively true breeding although fertility is low. This seems to indicate that gametes derived from cells with multivalent associations, or from those having allosyndetic bivalents, are inviable, or are at a selective disadvantage compared with those having the complete and balanced gene content of both species. A system promoting the formation of balanced gametes would therefore have considerable selective advantage, and it is such a system which exists in tetraploid wheat.

Geographical Distribution

Present areas of distribution of the possible diploid ancestors of tetraploid wheat do not necessarily indicate precisely where hybridization between the parental forms occurred. There may have been subsequent shifts in distribution, especially if hybridization followed by chromosome doubling occurred in relatively ancient time.

According to Eig[5] and Zhukovsky[41] *Aeg. speltoides* is widely distributed in Asia Minor, Syria and Palestine. This, according to Vavilov[39] is an area where wild forms of *T. monococcum*, *T. aegilopoides* and *T. thaoudar* are found. *T. dicoccoides*, Vavilov[39], also grows wild in this area and presumably originated here. Thus the evidence of contemporary distributions is not in conflict with the hypothesis that *Aeg. speltoides* contributed the *B* genome in the origin of polyploid wheat.

Discussion

The nature of the satellited chromosomes of tetraploid wheat and the discovery that both pairs are in the *B* genome dismisses the possibility that the tetraploids are autopolyploid derivatives of diploid wheat.

Karyotype analysis also indicates that *Agropyron triticeum* need not be considered as the possible source of *B*. In fact, studies of pairing at meiosis correctly interpreted, and results of breeding investigations indicate strongly that *Agropyron* did not contribute the *B* genome of wheat.

From the evidence of studies of karyotype, chromosome pairing in hybrids and geographical distribution, *Aegilops speltoides* fits most closely the requirements of the donor of the *B* genome of wheat. This supports the conclusion of Sarkar and Stebbins[26] from morphological studies. It might be speculated, therefore, that originally a form very like *Aeg. speltoides* crossed naturally with a diploid wheat growing in the same area. Natural doubling of the chromosome number would have resulted in the production of an amphiploid with low fertility resulting from frequent multivalents at meiosis. The original tetraploid would in fact, be very similar to the artificial amphiploid *T. monococcum* × *Aeg. speltoides* investigated in this study. A mechanism which

would restrict bivalent formation to identical chromosomes, that is, completely homologous chromosomes within each genome would have a pronounced selective advantage in giving improved fertility and genetical stability. It may well be that such a mechanism arose as a single mutation in the original amphiploid and became incorporated in tetraploid wheat.

It should also be indicated that *T. timopheevi* behaves very like *AB* tetraploids in hybrids with *Aeg. speltoides*. Further, it has similar satellited chromosomes, presumably in the *G* genome. Thus, apparently, the origin of the *AG* tetraploids is like that of the *AB*, and the differences between them have resulted from changes subsequent to their formation, as proposed by Sachs[27].

When the *D* genome of *Aeg. squarrosa* was added to the tetraploids in the origin of hexaploid wheat, the mechanism restricting pairing would prevent association of chromosomes of the *D* and those of the *A* and *B* genomes. If the diploidization mechanism had not already been present in the tetraploid parent, it is doubtful whether the hexaploid could have persisted as an annual form, because of meiotic irregularity.

It is interesting to consider how the hybrid combining the putative parents of hexaploid wheat would behave meiotically. Very likely, when the hybrid from crossing the amphiploid of diploid wheat × *Aeg. speltoides* with *Aeg. squarrosa* is obtained, pairing will be very similar to that in the 20-chromosome nullihaploid, studied by Riley[23], which is deficient for the gene restricting pairing. That is, pairing between homoeologous chromosomes in the three genomes should occur. The parallel hybrid with the *Aeg. bicornis*, instead of the *Aeg. speltoides* genome should give similar results since the gene restricting pairing is probably not present in *Aeg. bicornis*, although it is impossible to predict the exact interrelationships of this mechanism in related forms.

These suggestions about the origins of wheat have been prompted by the reappraisal of some old evidence in the light of some new information on the disposition of the satellited chromosomes, and on the control of meiosis. In the context of the new evidence apparently only *Aeg. speltoides* can qualify as Sarkar and Stebbins have already concluded from morphological evidence. If the polyploids have indeed originated in the way proposed, ideas on the phylogeny of wheat must be readjusted to associate the genera *Triticum* and *Aegilops* more closely together. Further, it would seem that the use of *Aegilops* as a source of variation in wheat improvement should be strongly urged, since there are already two doses of this genus in the common wheat of our fields.

Summary

It can be concluded from the analysis of karyotype, and chromosome pairing in hybrids, that *Aegilops speltoides* is the diploid species most closely approaching the requirements of the donor of the *B* genome of polyploid wheat. This sustains the conclusions of Sarkar and Stebbins based on morphological characters.

Support for this conclusion comes from two sources of evidence. First, that both pairs of satellited chromosomes of the polyploid wheats are in the *B* genome, and two similar pairs are found only in *Aeg. speltoides*. Secondly, a mechanism restricting intergenome pairing, has been discovered in the *B* genome of polyploid wheat and pairing data in hybrids has been reinterpreted in the light of this situation. This reappraisal shows that the chromosomes of *Aegilops speltoides* are closely related to those of the *B* genome.

Literature Cited

1. Anderson, E. Introgressive Hybridization. Wiley and Sons. New York 1949.

2. Bell, G. D. H. and L. Sachs. Investigations in the Triticinae. II. *Jour. Agric. Sci.* 43:105-115. 1953.

3. Bhatia, C. S. Cytology and genetics of some Indian Wheats. II. *Ann. Bot.* (N.S.) 2:335-371. 1938.

4. Camara, A. Chromosomes dos trigos hexaploides. *Agron. Lusit.* 6:221-251. 1944.

5. Eig, A. Monographisch—Kritische Übersicht der Grattung *Aegilops*. *Repert. nov. Spec. Regn. veg.* 55:1-228. 1929.

6. Jenkins, B. C. and A. Mochizuki. A new amphiploid from a cross between *Triticum durum* and *Agropyron elongatum* ($2n = 14$). *Wheat Inf. Service* 5:15. 1957.

7. Kihara, H. Uber cytologische Studien bei einigen Getreidiarten. I. *Bot. Mag. Tokyo* 32:17-38. 1919.

8. ———. Genomanalyse bei *Triticum* and *Aegilops*. I. *Cytologia* 2:106-156. 1930.

9. ———. Ein diplo-haploides Zwillingspaar bei *Triticum durum*. *Agr. and Hort.* 11:1425-1434. 1936.

10. ———. Die Entdeckung des DD—Analysators beim Weizen. *Agric. Hort. Japan* 19:889-890. 1944.

11. ———. Genomanalyse bei *Triticum* and *Aegilops*. IX. *Cytologia* 14:135-144. 1949.

12. ———, and F. A. Lilienfeld. A new synthesised 6x-wheat. *Proc. Eighth Int. Congr. Genet.* 1949.

13. Larson, R. E. Aneuploid analysis of inheritance of solid stem in common wheat. *Genetics* 37:597. 1952.

14. Levitzky, G. A., M. A. Sizara and V. A. Paddubnaja-Arnoldi. Comparative morphology of the chromosomes in wheat. *C.R. Acad. Sci. U.R.S.S.* 25:142-145. 1939.

15. Lilienfeld, F. A. H. Kihara: Genome analysis in *Triticum* and *Aegilops* X. *Cytologia* 16:101-123. 1951.

16. Matsumura, S. Genetics of some cereals. *Ann. Rep. Nat. Inst. Genet. Japan* 1:22-27. 1951.

17. McFadden, E. S. and E. R. Sears. The artificial synthesis of *Triticum spelta*. *Rec. Genet. Soc. Am.* 13:26-27. 1944. *Genetics* 30.

18. ———— and ————. The origin of *Triticum spelta* and its free-threshing hexaploid relatives. *Jour. Hered.* 37:81-89, 107-116. 1946.

19. ———— and ————. The genome approach in radical wheat breeding. *Jour. Amer. Soc. Agron.* 39:1011-1026. 1947.

20. MORRISON, J. W. Chromosome behavior in wheat monosomics. *Heredity* 7:203-217. 1953.

21. PATHAK, G. N. Studies in the cytology of cereals. *Jour. Genet.* 39:437-467. 1939.

22. PETO, F. H. Hybridization of *Triticum* and *Aegilops*. II. *Canad. Jour. Res.* C. 14:203-214. 1936.

23. RILEY, R. Chromosome pairing and haploids in wheat. *Proc. Tenth Int. Congr. Genet.* 1958.

24. ———— and V. CHAPMAN. Haploids and polyhaploids in *Aegilops* and *Triticum*. *Heredity* 11:195-207. 1957.

25. ROY, R. R. Cytogenetic investigations in intergeneric amphiploids between *Aegilops* and *Triticum*. *Disst. Univ. of Cambridge* 1-283. 1952.

26. SARKAR, P. and G. L. STEBBINS. Morphological evidence concerning the origin of the B genome in wheat. *Amer. Jour. Bot.* 43: 297-304. 1956.

27. SACHS, L. Chromosome behaviour in species hybrids with *Triticum timopheevi*. *Heredity* 7:49-58. 1953.

28. SAX, K. Sterility in wheat hybrids. II. *Genetics* 7:513-552. 1922.

29. SEARS, E. R. Chromosome pairing and fertility in hybrids and amphidiploids in the Triticinae. *Res. Bull. Mo. Agric. Exp. Sta.* 337:1-20. 1941.

30. ————. The cytology and genetics of the wheats and their relatives. *Advances in Genetics* 2:239-270. 1948.

31. ————. The *B* genome in wheat. *Wheat Inf. Service* 4:8-10. 1956.

32. ———— and M. OKAMOTO. Intergenomic chromosome relationships in hexaploid wheat. *Proc. Tenth Int. Cong. Genet.* 1958.

33. STEBBINS, G. L. and F. T. PUN. Artificial and natural hybrids in the Gramineae, Tribe Hordeae. VI. *Genetics* 38:600-608. 1953.

34. TANAKA, M. Chromosome pairing in hybrids between *Aegilops sharonensis* and some species of *Aegilops* and *Triticum*. *Wheat Inf. Service* 2:7-8. 1955.

35. ————. Chromosome pairing and fertility in the hybrid between the new amphidiploid S^b S^b *AA* and emmer wheat. *Wheat Inf. Service* 3:21-22. 1956.

36. THOMPSON, W. P. Chromosome homologies in wheat rye and *Aegilops*. *Canad. Jour. Res.* 4:624-634. 1931.

37. ————, E. J. BRITTEN and J. C. HARDING. The artificial synthesis of a 42-chromosome species resembling common wheat. *Canad. Jour. Res.* C. 21:134-144. 1943.

38. TJIO, J. H. and A. LEVAN. The use of oxyquinoline in chromosome analysis. *Ann. Estac. exp. Aula Dei.* 2:21-64. 1950.

39. VAVILOV, N. I. The origin, variation, immunity and breeding of cultivated plants. *Chron. Bot.* 13:1-364. 1949.

40. WAKAR, B. A. Cytologische Untersuchungen der ersten Generation der Weizen queckenbastarde. *Züchter.* 7:199-206. 1935.

41. ZHUKOVSKY, P. M. A critical-systematic survey of the species of the genus *Aegilops* L. *Bull. Appl. Bot. Genet. and Pl. Br.* 18:417-609. 1928.

42. OKAMOTO, M. Further information on identification of the chromosomes in the A and B genomes. *Wheat Inf. Service* 6:3-4. 1957.

LINKAGE STUDIES IN THE HONEY BEE

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ALTHOUGH many mutant types have been reported in honey bees (*Apis mellifera* L.), it is only recently that linkage has been studied to any extent. Laidlaw *et al.*[3] investigated the genetics of several eye-color mutants, and Rothenbuhler *et al.*[6] studied four recessive eye colors and a dominant wing mutant. In neither study was linkage found. Hachinohe and Onishi[1] determined a crossover value of 31 percent between the loci of a rudimentary wing mutant and a recessive lethal. Kerr and Laidlaw,[2] in their review of the genetics of bees, mentioned a close linkage between the loci of brick eye color and reduced facet number. In the present paper a new mutant and a number of linkage relationships are described.

In the honey bee, males (drones) develop from unfertilized eggs and, since they represent the gametes of their mother, can be used to establish segregation ratios. Females (queens and workers) develop from fertilized eggs and are diploid.

The new mutant, hairless (*h*), is a recessive causing reduced hairiness all over the body (Figure 2). The hair of newly emerged bees appears to be matted, and it soon rubs off so that the bees look almost completely hairless. Closer examination shows many hairs remaining in protected places. On the hind legs of worker bees the rows of bristles on the inside surfaces of the basitarsi and the spines of the pollen rakes on the tibiae are unaffected, but most of the long hairs that usually form the pollen basket are absent. Although hairless worker bees are highly viable, many hairless drones die before they emerge but reach sufficient maturity to be recognized as hairless. This mutant first appeared in a small percentage of the drone progeny of a heterozygous chartreuse queen.

Other recessive mutants used were cordovan body color (*cd*) and the eye colors of ivory (*i*), cream (*cr*), snow (*s*), chartreuse (*ch*), and brick (*bk*). A dominant mutant, Droopy wings (*D*), was also included in the tests. Several of the mutant stocks were provided by W. C. Rothenbuhler, Iowa State College, and H. H. Laidlaw, University of California (Davis). Ivory, cream, and snow cause a complete lack of pigment in eyes and are epistatic to chartreuse and brick. The chartreuse-brick phenotype is distinguishable from both chartreuse and brick. Droopy occurs only in the heterozygous condition because it is lethal in the haploid drone. The ivory stock was the same as the one used by Laidlaw *et al.*[3], and tests indicate identity with the ivory of Rothenbuhler *et al.*[6] The chartreuse used in these tests is apparently identical to the chartreuse of Rothenbuhler *et al.* It was also shown to be allelic to the chartreuse series of alleles described by Laidlaw *et al.*, but whether or not it is identical with one of them is not known.

WILD TYPE AND HAIRLESS WORKER BEES
Figure 2

The new mutant, hairless (*h*), shown in the lower photograph is a recessive causing reduced hairiness over entire body.

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Linkage Tests

Linkage tests between various pairs of these mutants were made with heterozygous queens which had inherited the two mutants from different parents. When the segregation ratio was to be determined in the drone progeny, the genetic constitution of the queen's mate was irrelevant. When it was to be determined in worker progeny, the queens were mated to mutant drones. The results are given in Table I.

Significant deviations from the 1:1:1:1 or 1:2:1 ratios expected with independent segregation were obtained in four of these tests. Although in test 1a the deviation was significant ($\chi^2 = 8.0$, P = 0.02-0.05), when the test was repeated (1b) with worker progeny and with much larger numbers, no significant deviation was found ($\chi^2 = 5.0$, P = 0.1-0.2). A conclusion of linkage between cordovan and hairless is, therefore, not justified.

In test 9 a close linkage between hairless and chartreuse is indicated, but the crossover classes (chartreuse-hairless and wild-type) deviate significantly from the expected 1:1 ratio ($\chi^2 = 5.68$, P = 0.01-0.02). Since some of the hairless individuals that die before emergence are difficult to classify as to eye color, the hairless class has probably been increased at the expense of the chartreuse-hairless class. Furthermore, since the viability of chartreuse is high, as can be seen from test 4, the best estimate of crossing-over is obtained from the chartreuse and wild-type classes and amounts to 4.1 percent.

Tests 14a and 14b show considerable deviation from the expected 1:2:1 ratio. If this deviation was caused by linkage of brick and snow, the wild-type and combined snow and brick-snow classes should be reduced. This is the case in test 14a but in test 14b it is the brick and combined snow and brick-snow classes that are reduced. If the deviation was caused by low viability of brick then the brick and combined snow and brick-snow classes should be reduced in both tests. The data can be explained if one assumes a semi-lethal closely linked with the brick locus which might be dominant or recessive. In test 14a it was inherited with the maternal wild-type allele of brick while in test 14b it was inherited with brick from the queen parent if dominant or from both the queen and some, or all, of the drone parents (multiple drone mating) if recessive, and was homozygous or not present in the other brick linkage tests in Table I. The situation is complicated by the possibility that a similar semi-lethal is associated with the snow locus as will be pointed out in the discussion of Table II. The following facts give additional support to this explanation: In a test of viability of brick a heterozygous queen produced 482 brick and 489 wild-type drones as if the semi-lethal were homozygous or absent, while another produced 321 brick and 391 wild type, a significant deviation from the ex-

TABLE I. Linkage relationships of visible mutants in the honey bee

Test No.	Genotype of parents: Queen	Genotype of parents: Drone	Type of progeny	Phenotypes and frequencies of progenies‡				χ^2
1a	*cd*;*h*$^+$/*cd*$^+$;*h*	†	Drone	294 *cd*;	236 *h*;	262 *cd-h*;	289+	8.0*
1b	*cd*;*h*$^+$/*cd*$^+$;*h*	*cd*; *h*	Worker	2168 *cd*;	2253 *h*;	2226 *cd-h*;	2316+	5.0
2	*cd*;*i*$^+$/*cd*$^+$;*i*	†	Drone	1709 *cd*;	1717 *i*;	1687 *cd-i*;	1683+	0.5
3	*cd*;*bk*$^+$/*cd*$^+$;*bk*	†	Drone	487 *cd*;	524 *bk*;	528 *cd-bk*;	540+	3.0
4	*cd*;*ch*$^+$/*cd*$^+$;*ch*	†	Drone	378 *cd*;	394 *ch*;	382 *cd-ch*;	374+	0.6
5	*cd*;*s*$^+$/*cd*$^+$;*s*	†	Drone	673 *cd*;	646 *s*;	687 *cd-s*;	672+	1.3
6	*cd*;*cr*$^+$/*cd*$^+$;*cr*	†	Drone	633 *cd*;	669 *cr*;	658 *cd-cr*;	665+	1.2
7	*h*;*i*$^+$/*h*$^+$;*i*	†	Drone	173 *h*;	157 *i*;	175 *h-i*;	163+	1.3
8	*h*;*bk*$^+$/*h*$^+$;*bk*	†	Drone	441 *h*;	421 *bk*;	403 *bk-h*;	421+	1.7
9	*h*;*ch*$^+$/*h*$^+$;*ch*	†	Drone	3131 *h*;	3064 *ch*;	96 *ch-h*;	132+	5544.0§
10	*h*;*s*$^+$/*h*$^+$;*s*	†	Drone	333 *h*;	350 *s*;	372 *h-s*;	324+	3.9
11	*h*;*cr*$^+$/*h*$^+$;*cr*	†	Drone	283 *h*;	296 *cr*;	279 *h-cr*;	311+	2.1
12	*i*;*bk*$^+$/*i*$^+$;*bk*	†	Drone	305 *bk*;	587 *i* (and *bk-i*);		333+	3.4
13	*i*;*ch*$^+$/*i*$^+$;*ch*	†	Drone	283 *ch*;	552 *i* (and *i-ch*);		273+	0.2
14a	*bk*;*s*$^+$/*bk*$^+$;*s*	†	Drone	345 *bk*;	562 *s* (and *bk-s*);		284+	10.0§
14b	*bk*;*s*$^+$/*bk*$^+$;*s*	*bk*;*s*	Worker	1751 *bk*;	3850 *s* (and *bk-s*);		2140+	39.3§
15	*bk*;*cr*$^+$/*bk*$^+$;*cr*	†	Drone	702 *bk*;	1399 *cr* (and *bk-cr*);		768+	4.8
16	*D*;*cd*$^+$/*D*$^+$;*cd*	*D*$^+$;*cd*	Worker	716 *D*;	697 *cd*;	768 *D-cd*;	724+	3.7
17	*D*;*h*$^+$/*D*$^+$;*h*	*D*$^+$;*h*	Worker	281 *D*;	270 *h*;	273 *D-h*;	298+	1.7
18	*D*;*bk*$^+$/*D*$^+$;*bk*	*D*$^+$;*bk*	Worker	235 *D*;	245 *bk*;	228 *D-bk*;	236+	0.6

*Significant deviation from random assortment (P < 0.05)

§Highly significant deviation from random assortment (P < 0.01).

†Genetic constitution irrelevant.

‡Where parentheses are used, the phenotype is indicated by the gene symbol outside the parentheses.

pected 1:1 ratio ($\chi^2 = 6.9$, $P = <0.01$), which suggests the presence of the semi-lethal on the brick chromosome. More critical experiments with viability studies are needed to determine the correctness of this explanation, and the characteristics of the semi-lethal.

All the visible mutants were tested for possible linkage with the lethal alleles described by the writer[4], which have now been shown by Rothenbuhler[5], to determine sex as in *Habrobracon*[7] and will hereafter be referred to as sex alleles. The members of this series act in such a way that heterozygotes are females and hemizygotes and homozygotes are males. In *Habrobracon* most of the homozygotes die as eggs, but a few live to maturity. In the honey bee they all die as eggs; however, small patches of diploid male tissue can survive in mosaic individuals.

Linkage tests between the sex alleles and recessives were made by crossing homozygous mutant queens to wild-type drones, followed by several brother-sister individual matings, and then determining the mutant to wild-type ratio in low-hatch progenies. In tests with the dominant mutant Droopy, which occurs only as a heterozygote, Droopy queens were mated individually to their wild-type brothers and the ratios of Droopy to wild-type workers determined in low-hatch progenies. In both types of tests the mutant segregated independently of the sex alleles if half the members of low-hatch progenies were mutant and the other half were wild-type. Any other ratio would indicate linkage, and the proportion of the two types of offspring would measure the degree of linkage. To establish the viability of the visible mutants, the ratio of mutant to wild-type individuals was also determined in one or more high-hatch progenies involving each mutant. The results are given in Table II; no linkage was indicated.

TABLE II. Linkage relationships of visible mutants to sex alleles in the honey bee

Mutant	Hatchability of progenies	Number of worker progeny		χ^2
		Mutant	Wild-type	
ivory	Low	1972	2031	0.9
	High	2056	2024	0.2
cream	Low	597	602	0.02
		667	641	0.1
	High	508	490	0.5
		475	535	3.6
snow	Low	546	558	0.1
		442	510	4.8*
	High	466	476	0.1
brick	Low	737	802	2.7
	High	1000	1154	11.0†
chartreuse	Low	732	744	0.1
		1168	1219	1.1
	High	1548	1614	1.4
cordovan	Low	1376	1363	0.06
	High	1234	1276	0.7
hairless	Low	304	277	1.2
		984	940	1.0
	High	1678	1597	0.1
droopy	Low	666	620	1.6
		623	619	0.01
		514	530	0.2
	High	1013	1042	0.4
		1095	1034	1.7

*Significant deviation from expected 1:1 ratio ($P < 0.05$).

†Highly significant deviation from expected 1:1 ratio ($P < 0.01$).

The deviation from random segregation in the high-hatch progeny of the brick test might have been caused by the semi-lethal already discussed in connection with the results of tests 14a and 14b of Table I. In this test the semi-lethal was linked with the brick locus and carried in the maternal brick chromosome if dominant or in the brick chromosomes of both parents if recessive. The deviation in one of the low-hatch progenies of the snow test might be similarly explained. It is not considered to indicate linkage because in the other low-hatch progeny a good 1:1 ratio was obtained.

The results reported in this paper combined with those of Laidlaw *et al.*[3] and Rothenbuhler *et al.*[6] include all the possible 2-point combinations between the genes studied. Although the total data establish linkage only between hairless and chartreuse, a loose linkage might exist among some of the other mutants and escape detection because of the lack of appropriate genetic markers.

Literature Cited

1. Hachinohe, Y. and N. Onishi. On the new mutation "rudimental wing" in the honeybee (*Apis mellifica* L.). In Japanese, English summary, *Bull. Nat. Inst. Agric. Sci., Ser.* G, 7:139-145. 1953.

2. Kerr, W. E. and H. H. Laidlaw. Genetics of bees. *Advances in Genetics* M. Demerec, ed., Academic Press, New York. Vol. 8, pp. 109-153. 1956.

3. Laidlaw, H. H., M. M. Green and W. E. Kerr. Genetics of several eye color mutants in the honey bee (*Apis mellifera* L.). *Jour. Hered.* 44:246-250. 1953.

4. Mackensen, O. Viability and sex determination in the honey bee (*Apis mellifera* L.). *Genetics* 36:500-509. 1951.

5. Rothenbuhler, Walter C. Diploid

male tissue as new evidence on sex determination in honey bees. *Jour. Hered.* 48:160-168. 1957.

6. ————, JOHN W. GOWEN and O. W. PARK. Allelic and linkage relationships of five mutant genes in honey bees (*Apis mellifera* L.). *Jour. Hered.* 44:251-253. 1953.

7. WHITING, P. W. Multiple alleles in complementary sex determination in *Habrobracon*. *Genetics* 28:365-382. 1943.

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SEX-REVERSAL AND ABNORMAL SEX RATIOS IN THE DOMESTIC FOWL

H. R. CARNE AND W. L. WATERHOUSE*

THE opportunity has been presented to us of making the following observations on apparent sex-reversal and abnormal sex ratios in the fowl.

One bird of what appeared to be a normal pen of Black Orpington pullets showed reversal of sex, eventually developing into a normal-looking and aggressive cock. The birds were purchased as six-week-old (July hatched) pullets from a reputable poultry farmer. In July the following year, when some of the birds were laying, it was noticed that one of them appeared to be "off color." It subsequently recovered and the comb was then observed to increase in size and male plumage began to develop. It was at about 13 to 14 months from hatching that the male characteristics were clearly marked, and about a month later the bird began to behave as a male. There was no definite evidence that this bird had ever laid eggs. It was then mated with a pen of Black Orpington pullets. A number of clutches of eggs were placed under hens, but there was a very heavy mortality in the shell, and a total of only 26 chicks was hatched, of which every one showed typical external male characteristics on development.

The bird was then mated with three virgin Australorp pullets. These birds laid respectively 63, 35 and 45 eggs during the period of the experiment and all were incubated. There was a high percentage of infertile eggs and also a large number of embryos died in the shell, most during the last week of incubation. However, it was possible to determine the sex by dissection in 24 instances of chicks which either hatched or were sufficiently advanced at the time of death in the shell for their sex to be determined. Details are given in Table I.

Every chick whose sex could be determined was also male, so that out of a total of 50 offspring of this cock, every one was a male.

The three pullets used in this experiment were subsequently mated with a White Leghorn cockerel and showed a normal proportion of male and female chicks in subsequent hatches.

Post mortem examination of the Black Orpington cock revealed a pair of normal, well developed testes, no sign of any ovarian or other female tissues, but the right kidney was absent.

It is difficult in the absence of further evidence to put forward an explanation which carries any conviction. If we suppose that the absence of females is due to both X chromosomes of the cock carrying a lethal, we must further suppose that the two lethal genes are closely linked or included in a pericentric in-

(*Continued on Page* 106)

TABLE I. Details of the experiment

Hen no.	Eggs incubated	Infertile eggs	Fertile eggs			Chicks or embryo sexed*
		Number	Number	Died	Hatched	
40	63	22	41	40	1	12
50	35	16	19	19	0	3
76	45	13	32	32	0	9

*All males.

*Department of Veterinary Pathology and Bacteriology, University of Sydney; and 30 Chelmsford Avenue, Lindfield, N.S.W. Australia, respectively. We are indebted to Dr. F. H. W. Morley and Dr. J. M. Rendel for suggesting possible mechanisms to account for these observations.

HAPLOIDS OF THE COMMON POTATO

R. W. Hougas, S. J. Peloquin and R. W. Ross*

INDUCED polyploidy, particularly since the discovery of the use of colchicine, is well recognized as a valuable tool for plant breeding[10]. The colchicine technique has resulted in the production of innumerable polyploid plants[2].

No such efficient method for the reverse process of reduction in chromosome numbers of polyploid plants has been found. Haploid plants, however, have been reported rarely among such polyploid plants as tobacco, wheat, cotton, alfalfa, smooth bromegrass and certain of the *Solanum* species. Such haploid plants, supposedly arising through parthenogenesis, have been observed (1) among seedling progeny of interspecific and intergeneric matings and (2) among normal populations of seedling plants either as individual plants or as one member of multiple seedlings.

The common potato, *Solanum tuberosum* L., is considered to be an autotetraploid by most investigators. It has, however, also been suggested that it could be a segmental allopolyploid. Haploid plants of this species would seem to offer material of unusual promise for both potato breeding and genetic and cytogenetic researches.

Experimental Procedure and Results

Eight haploid *Solanum demissum* plants ($2n=36$) were found in 1953 at the Potato Introduction Station, Sturgeon Bay, Wisconsin, among 168 offspring resulting from matings of the Mexican hexaploid *Solanum demissum* ($2n=72$) with 13 diploid *Solanum* species ($2n=24$). The occurrence of *S. demissum* haploids in such frequency suggested the possibility of obtaining haploid plants of other polyploid *Solanum* species through a similar system of interspecific matings. Accordingly a series of tetraploid (♀)—diploid (♂) matings of the tuber-bearing *Solanum* species were undertaken in the summer of 1956 at this Station.

Ten selections of the common tetraploid potato, $2n=48$, (Minn. 1, Minn. 10-5-12, Minn. 15-2-10-1-2, Minn. 20-20-34, Minn. 44-2, Minn 113-1, Minn. 113-6, ND 457-1, ND 457-1-35, and Katahdin) were used as pistillate parents in matings with seven diploid selections (*S. phureja*-P.I. 205561, P.I. 225682; *S. simplicifolium*—P.I. 208866; *S. verrucosum*—P.I. 161128; *S.* spp.—P.I. 217453; *S. phureja* F_1—WRF 173.7, WRF 175.4). An attempt was made to select parental stocks with markedly contrasting genetic characters so that the phenotype of the F_1 hybrids would be distinct from that of any haploids occurring through maternal parthenogenesis. The characters used included leaf morphology and pigmentation of various plant parts such as flower, stem and tuber.

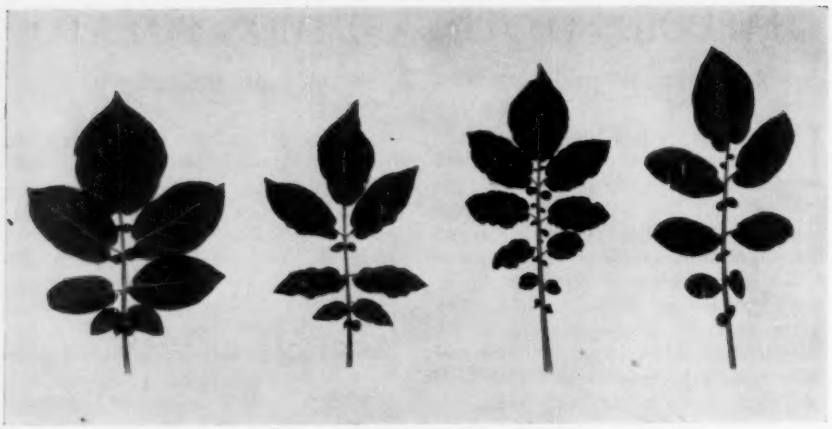
Fruit set was erratic and fruit development was considerably retarded following these matings. Only a few fruits approached normal development and many dropped when one-fourth normal size or less.

A total of 959 plump seeds was obtained from 6041 pollinations involving 37 different parental combinations. The average number of plump seeds per fruit was extremely low, ranging from zero to slightly more than two for the matings attempted. Eight-hundred and six of the 959 seeds collected were viable but several of the seedlings died in the pretransplant stage. Loss of individuals in the post-transplant stage was low.

The search for haploid individuals among the resulting seedlings was conducted under the premise that:

1. The frequency of haploid plants would probably be very low.

*Crops Research Division, ARS, USDA, and the University of Wisconsin in Cooperation with the Inter-Regional Potato Introduction (IR-1) Project.



LEAVES FROM NORMAL AND HAPLOID PLANTS

Figure 3

From left to right are shown the variety Katahdin and haploids US-W1, US-W6 and US W12.

2. The haploid plants should resemble the pistillate parent but could be somewhat reduced in overall size.
3. The haploids should lack all phenotypic characters of the F_1 hybrids conditioned by the pollen parent.
4. The haploid plants would lack the heterosis of the hybrid offspring.

The seedling populations were scrutinized daily for possible haploids. The chromosome number of such plants was then ascertained by use of the aceto-carmine, root-tip smear method.

Twenty-eight haploid plants ($2n=24$) of *S. tuberosum* were found among the seedling families resulting from the $4x$-$2x$ interspecific matings. One of these haploids has been previously reported[5]. One or more haploids were obtained from seven of the 10 *S. tuberosum* selections used as pistillate parents (Table I). The suppositions made, prior to the experiment, concerning the frequency, appearance and vigor of the haploids were fulfilled rather closely with one seeming exception, the frequency of haploids. A haploid frequency rate of one plant in 28.8, as based on the total viable offspring, appears surprisingly high. It should be pointed out, however, that based upon the total pollinations (6041), the total offspring realized in this trial is extremely low. A conservative estimate of the total viable offspring resulting from a similar number of successful intraspecific matings of the pistillate parent would be 150,000 to 300,000. Hence the measure of haploid frequency based upon the total offspring of this trial must take into consideration that viable F_1 hybrids occur rather infrequently in the matings attempted.

Morphologically the haploid plants were smaller and less vigorous than the

TABLE I. Frequencies of haploids in *Solanum tuberosum*

Pistillate parent	Flowers pollinated	Seeds	Seeds germinated	Haploids
Minn. 1	292	111	89	0
Minn. 10-5-12	225	19	14	1
Minn. 15-2-10-1-2	731	126	116	1
Minn. 20-20-34	204	116	108	2
Minn. 44-2	280	62	47	1
Minn. 113-1	210	104	100	1
Minn. 113-6	138	51	41	0
ND 457-1	157	89	80	1
ND 457-1-35	102	7	3	0
Katahdin	3702	274	208	21
TOTAL	6041	959	806	28

F_1 hybrids as well as the *S. tuberosum* selection from which they originated. The stomata and pollen grains of the haploids were smaller than those of either the F_1 hybrids or the *S. tuberosum* selections, but approximately the same size as the stomata and pollen grains of diploid species such as *S. phureja*. The leaflets of the haploids were small and narrow in contrast to the large, broad leaflets characteristic of the *S. tuberosum* selections (Figure 3). There was considerable variation among haploid sibs of the *S. tuberosum* variety Katahdin, but this is not unexpected in view of the considerable heterozygosity characteristic of commercial potato varieties[7] and the increased potential for expression of variability resulting from the reduction in ploidy[10].

Considering the large numbers of seedlings grown annually by the potato breeders of this nation and abroad, it seems probable that haploid plants occur from time to time but are lost in the normal sequence of procedures of breeding for commercial varieties (propagation, screening and selection). Since the common potato is known to be highly heterozygous[6,8], the variation in seedling populations due to segregation complicates the task of selecting possible haploids. The practicability of searching for haploids in natural populations depends initially upon trustworthy criteria for the macroscopic recognition of such plants. Otherwise the search would be both laborious and time-consuming since it would necessitate selection, for subsequent microscopic examination, of all the smaller, less vigorous plants from the population. Twenty-eight natural seedling populations of the common potato, involving more than three thousand plants, were grown to the transplant stage (1½ to 2½ inches) and examined, but no haploids were recognized.

Haploid plants have been found, arising from one member of polyembryonic seeds, in several species[11]. Harland[4] found an unusually high haploid frequency among twin seedlings of Sea Island cotton (*Gossypium barbadense* L.). Morgan and Rappleye[9] also report a high frequency of haploids among twin seedlings of certain pepper varieties.

The results of the cotton and pepper studies suggested that haploids might occur among twin seedlings of potato. A search for twin seedlings was therefore made in natural seedling populations of the common potato. Eleven pairs of twins were found among 20,000 seedling plants. None of the plants obtained from twin seedlings in these trials were haploids.

Discussion

The primary purpose of this experiment was to determine whether interspecific matings could be used as a tool for obtaining haploids of the polyploid *Solanum* species. Several factors, such as time of pollination[3] and choice of both male and female parents[1], are known to affect haploid frequency in other plants. With this knowledge, the desirability of planning the experiment in such a fashion as to incorporate rather broad samplings of these factors was obvious. It was equally apparent that practical limitations of plant materials and labor precluded the possibility of preparing an experimental design for the initial exploratory trial which would permit critical analysis of the effect of these factors on haploid frequency. Consequently, although the primary objective of the trial was realized, that of obtaining *Solanum* haploids through interspecific matings, the evidence was not conclusive for individual factors influencing haploid frequency.

Numerous haploids are essential if the full potentiality of such materials for basic researches in genetics, cytogenetics and potato breeding is to be realized. Reasonable reliability in macroscopic identification of haploids in natural seedling populations, especially in the post-transplant stage of growth, may indeed be feasible now that morphological descriptions of several *S. tuberosum* haploid plants are available. However, some means of markedly increasing the frequency of haploidy is essential if stockpiles of haploids are to be accumulated expeditiously. Consequently studies are currently being conducted to investigate the effect of several factors on haploid frequency. The frequency of haploids in maize is affected by the choice of both the pistillate and the staminate "parent"[1]. Therefore a survey of parental material, especially the staminate "parent," seems a promising initial approach.

Summary

Interspecific matings were used as a tool for obtaining haploids of *S. tuberosum*. Ten selections of the commercial potato ($2n=48$) were used as pistillate parents in matings with seven diploid selections ($2n=24$). Stocks with contrasting genetic characters, such as pigmentation and leaf morphology, were selected

in order to distinguish the F_1 hybrids from the haploids. Eight-hundred and six seedlings were obtained from 6041 pollinations. Twenty-eight haploid plants ($2n=24$) of *S. tuberosum* were found among these seedlings. Morphologically the haploid plants were smaller than either the F_1 hybrids or the *S. tuberosum* selection from which they originated. Large numbers of haploids will be needed if their potential for researches in genetics, cytogenetics and potato breeding is to be realized.

Literature Cited

1. Chase, S. S. Monoploids in maize. In: Heterosis (ed. by J. W. Gowen) Iowa State College Press, Ames, Iowa. 1952.

2. Eigsti, O. J. and P. Dustin. Colchicine. Iowa State College Press, Ames, Iowa. 1955.

3. Gerrish, E. E. Studies of the monoploid method of producing homozygous diploids in *Zea mays*. *Dissertation Abstrs.* 16:2285-2286. 1956.

4. Harland, S. C. Haploids in polyembryonic seeds of Sea Island cotton. *Jour. Hered.* 27:229-31 1936.

5. Hougas, R. W. and S. J. Peloquin. A haploid plant of the potato variety Katahdin. *Nature* 180:1209-1210. 1957.

6. Krantz, F. A. Potato breeding methods. III. A suggested procedure for potato breeding. *Minn. Agr. Exp. Sta. Tech. Bul.* 173:24. 1946.

7. ————. Potato breeding in the United States. *Zeit. Pflanzenzücht.* 29:388-93. 1951.

8. Lunden, A. P. Arvelighetsundersokelser i potet. *Meld Norges Landbruk.* 20:1-156. 1937.

9. Morgan, D. T., Jr. and R. D. Rappleye. Twin and triplet pepper seedlings. *Jour. Hered.* 41:91-95. 1950.

10. Stebbins, G. L. Artificial polyploidy as a tool in plant breeding. *Brookhaven Symposia in Biology* 9:37-50. 1956.

11. Webber, J. M. Polyembryony. *Bot. Rev.* 6:575-598. 1940.

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Sex Reversal and Abnormal Sex Ratios in the Domestic Fowl

(*Continued from Page* 102)

version, for which the cock is heterozygous, otherwise crossing-over would result in some X chromosomes carrying no lethal and none were found. To account for the lethal effect in males the genes must be supposed dominant and this makes it very hard to account for the cock himself as he would then be supposed to carry two dominant lethals, one on each X, and yet survive, so these two lethals would have to compensate one another in some way; the whole explanation becomes very improbable. It is possible that a translocation of a piece of one X into the other took place at an early cleavage division; this would not disturb the cock himself as he would still have a normal chromosome complement in each cell; his gametes, however, would carry either a deficiency or a duplication which would have to be sufficiently disturbing to be lethal in homozygous condition at an early stage and in heterozygous condition at or about hatching.

We were unable to make matings to test these hypotheses, but wish to record an event of interest to animal geneticists.

STUDIES ON THE INHERITANCE OF COLOBOMA OF THE IRIS IN THE DOMESTIC FOWL

F. H. WILCOX*

A DEFECTIVE formation of the eye, characterized by an indentation of the iris and subsequently referred to as coloboma, has been observed in chickens but with a low frequency. No evidence has been reported in the literature suggesting a genetic influence, although such evidence has been found for mammals[4]. Landauer[3] and Gayer[2] observed coloboma in chick embryos homozygous for the Creeper gene, but this was probably different from the condition observed in the present study since more than the iris was affected.

In 1953 three females and one male from the same sire were found to have coloboma in one eye. Each of these females was closely related to a hen hatched the previous year, which also had coloboma. Since these findings suggested a genetic influence, matings were designed to test this.

Experimental Matings

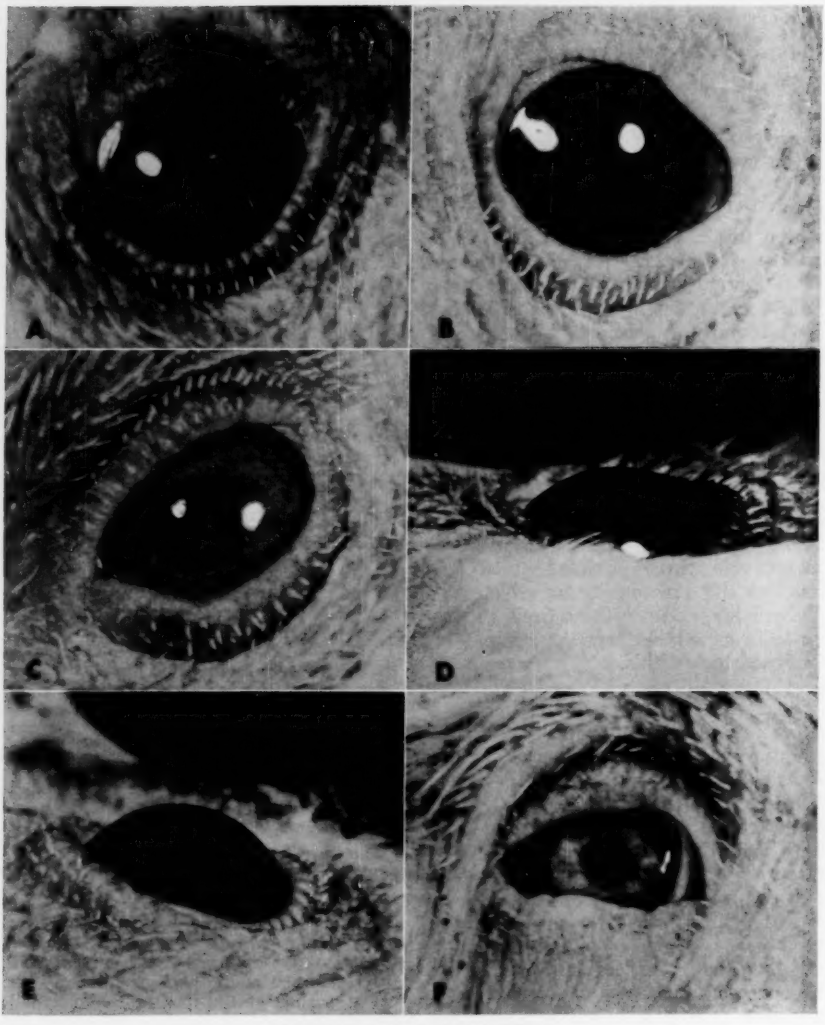
The male with coloboma was mated to four females with coloboma, to three females without coloboma but whose dam had coloboma, and to four females without coloboma but with one sister with coloboma. All dams used were closely related. The chicks from these matings were classified for coloboma in bright light at six weeks of age. An attempt was made to classify them at hatching, but considerable difficulty was encountered due to the dark color of the iris. By six weeks of age sufficient yellow pigment had been deposited in the iris for classification. Both eyes of each chicken were classified for presence and absence as well as degree of coloboma and for any other ocular anomalies that were present. From these progeny two males and five females were selected from families with a high incidence of coloboma, and were mated together. All of these birds had coloboma in one eye, except for one male with bilateral coloboma. The progeny from these matings were classified as previously described.

Results

The results of classification of the first and second generation chicks are given in Table I. Although the total incidence of coloboma increased from 22.0 percent to 27.9 percent from the first to second generation, these percentages are not significantly different ($P = 0.50\text{-}0.25$). When the chicks of the first generation are arranged according to the three classes of their dams (Table II), it can be seen that the chicks from dams with coloboma showed a higher incidence than chicks from normal dams. It is apparent that one generation of sib testing did not materially affect the incidence of coloboma, which is evident when a comparison is made of the incidence of coloboma in chicks from dams with coloboma in both generations. Table I shows there was little apparent difference in the incidence of coloboma in the right versus the left eye. Males had a much lower incidence of coloboma in both generations ($P = <0.01$).

The majority of eyes observed were classified as normal (Figure 4*A*), and possessed a round pupil in the center of the iris. Most of those with coloboma of the iris appeared as the one shown in Figure 4*B*, with an indentation of the pupil into the iris in the ventral end of the iris. The defect was much more obvious when observed in bright light, in which the iris was maximally constricted. Occasionally a coloboma was observed where the indentation of the pupil was towards the dorsal portion of the iris, (see Figure 4*C*). Several cases of pronounced bulging of the eye were observed in this stock. A normal eye from another strain of White Leghorns is shown in Figure 4*D*, and an eye with bulging is shown in 4*E*. In one case bulging developed in an eye previously classified as coloboma. Another abnormality observed oc-

*Departments of Poultry Husbandry at the University of Maryland, College Park, Maryland, and at Cornell University, Ithaca, New York. Scientific article No. A662. Contribution No. 2861 of the Maryland Agricultural Experiment Station, Department of Poultry Husbandry. The author is indebted to Dr. R. K. Cole for making the stock available.



OCULAR ABNORMALITIES

Figure 4

Photographs of ocular abnormalities observed in White Leghorns with a high incidence of coloboma. *A*—Eye classified as normal. *B*—Eye classified as coloboma. This type was observed the most frequently. *C*—Eye classified as coloboma. This type was observed infrequently. *D*—Outline of normal eye in a White Leghorn from another strain. *E*—Outline of eye classified as bulging. *F*—Eye classified as sunken.

casionally int his stock was a sunken eye, (Figure 4*F*), which was reduced considerably in size. This defect was not similar to ocular lymphomatosis, since the entire eye was markedly affected. In one case this abnormality occurred in an eye previously classified as bulging.

Birds with coloboma appeared to be normal in all other respects. There was no difference in iris color, and the irises dilated and constricted normally. Dissection and macroscopic observation of the other portions of affected eyes revealed no other anomalies besides those mentioned. Reproductive performance was normal.

Discussion

The results of these matings provide evidence that coloboma of the iris is an inherited abnormality in the domestic fowl. They can be explained equally well by the action of genes at several loci, or at one locus but with incomplete penetrance. The latter possibility is strengthened by the fact that in most cases only one eye was affected yet presumably both eyes had the same genotype. In view of this apparent low penetrance, it will be difficult to determine the exact mode of inheritance.

The noticeable bulging of a few eyes in this line selected for coloboma suggests that pressure inside the eye has increased. Such pressure changes might also be involved in formation of the coloboma itself. Coulombre[1] has shown intraocular pressure to be of importance in normal development of the chick eye. Further evidence on this is that the eyes of the coloboma stock described here weighed more than those of another strain of White Leghorns. These differences could, however, be the result of strain differences unrelated to coloboma. The average percentages of eye weight of total body weight at nine weeks of age were 0.305 and 0.338 percent for four males and one female, respectively, of the coloboma line, whereas they were 0.257 and 0.276 percent for two males and one female, respectively, of another strain of White Leghorns. Although such was not observed, it appears likely that increased pressure caused the sunken eye condition by a rupture of the eye wall. Since this would involve loss of sight, poultrymen conducting a breeding program should eliminate birds with coloboma from their flock. Such a procedure would be advisable since (1) coloboma is inherited and (2) there is some evidence of a relationship between coloboma and the sunken eye condition.

Summary

Closely related White Leghorn hens with coloboma of one of their irises or that had dams or sisters with this defect were mated to a male with coloboma. Classification of chicks from these matings at six weeks of age showed 22.0 percent with coloboma of one or both irises. Those with coloboma from families with a high incidence were then mated together, and an incidence of 27.9

TABLE I. Incidence of coloboma of the iris of first and second generation progeny when classified at six weeks of age

	Males		Females		Both sexes	
First generation	No.	%	No.	%	No.	%
No. chicks classified	60		81		141	
Unilateral, left	3	5.0	9	11.1	12	8.5
Unilateral, right	4	6.7	14	17.3	18	12.8
Bilateral	1	1.7	0	0.0	1	0.7
Total incidence	8	13.3	23	28.4	31	22.0
Second generation						
No. chicks classified	24		37		61	
Unilateral, left	4	16.7	5	13.5	9	14.8
Unilateral, right	0	0.0	4	10.8	4	6.6
Bilateral	0	0.0	4	10.8	4	6.6
Total incidence	4	16.7	13	35.1	17	27.9

TABLE II. Incidence of coloboma of the iris of first generation progeny of a single sire when classified at six weeks of age and grouped according to phenotypes of ancestors

Grouping according to ancestry	No. with coloboma	No. with normal eye	Percent coloboma
From dam with coloboma	32	12	27.3
From normal dam, but maternal grand-dam with coloboma	49	14	22.2
From normal dam, but one of dam's sisters with coloboma	29	5	17.2

percent was observed in their progeny. The incidence of coloboma was twice as great in females as in males. Bulging eye and sunken eye were also observed in this stock. The results obtained have provided evidence that coloboma of the iris is inherited, and it is suggested that an undetermined number of genes are operating with low penetrance.

Literature Cited

1. COULOMBRE, ALFRED J. The role of intraocular pressure in the development of the chick eye. I. Control of eye size. *Jour. Exp. Zool.* 133:211-226. 1956.

2. GAYER, KENNETH. A study of coloboma and other abnormalities in transplants of eye primordia from normal and Creeper chick embryos. *Jour. Exp. Zool.* 89:103-133. 1942.

3. LANDAUER, WALTER. Untersuchungen uber das Kruperhuhn. IV. Die Missbildungen homozygoter Kruperembryonen auf spateren Entwicklungsstadien. *Z. mikroskop.-anat. Forsch.* 32:359-412. 1933.

4. MANN, IDA. Developmental Abnormalities of the Eye. Cambridge University Press. 1937.

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A MODIFICATION OF THE "DOUBLE-CROSS" TECHNIQUE OF BREEDING HYBRID POULTRY

G. B. RITCHIE*

IT is known that the first cross or "F_1" between certain inbred lines of poultry give egg yields significantly and substantially higher than those from outbred stock of the same breed[1, 2].

In the United States "hybrid" or "incross-bred" hens are available for commercial production and appear to be successful; to what extent this depends upon fashion, advertising and the prestige of hybrid maize, is uncertain. This method, however, might give greater production, less variability and earlier maturity than do more conventional methods of breeding.

One method of hybrid breeding in commercial use is apparently the "double-cross" technique. This involves four inbred lines which are mated together in pairs to give two "F_1's," which are mated together to give a so-called "double-cross" generation. The method was designed for hybrid maize, to lower the cost of production of commercial hybrid seed. It offers no major genetical advantage over the plain "F_1", and is in fact likely to be less uniform and to have a slightly lower yield. Almost the only justification of the double-cross is where the inbred lines have such low viability or yield that the price of "F_1" hatching eggs is uneconomic for commercial producers.

The production of commercial double-cross birds involves at least seven different matings every season: that is the perpetuation of the four inbred lines, the production of the two "F_1's", and the production of the double-cross generation itself. Below are particulars of a similar cross involving only three different matings.

First consider the behavior of a single locus in the double-cross, assuming it to be homozygous in each of the four lines, and giving the allele present in each line a different designation.

Line A	Line B	Line C	Line D
$^aA\ ^aA$	$^bB\ ^bB$	$^cC\ ^cC$	$^dD\ ^dD$

Cross line A with line B, and C with D. There are two "F_1's":

F_1 $(A \times B)$	F_1 $(C \times D)$
$^aA\ ^bB$	$^cC\ ^dD$

On crossing the F_1's the double-cross is produced, having a mixture of four genotypes with regard to this locus:

Double-cross $[(A \times B) \times (C \times D)]$

$\frac{1}{4}{}^aA\ ^cC$, $\frac{1}{4}{}^aA\ ^dD$, $\frac{1}{4}{}^bB\ ^cC$, $\frac{1}{4}{}^bB\ ^dD$.

Now let us consider the composition of the F_2's from the above F_1's; they are:

F_2 $(A \times B)$

$\frac{1}{4}{}^aA\ ^aA$, $\frac{1}{2}{}^aA\ ^bB$, $\frac{1}{4}{}^bB\ ^bB$.

$(F_2$ $(C \times D)$

$\frac{1}{4}{}^cC\ ^cC$, $\frac{1}{2}{}^cC\ ^dD$, $\frac{1}{4}{}^dD\ ^dD$.

If these F_2's are crossed the resulting generation has the composition:

$F_2 (A \times B) \times F_2 (C \times D)$

$\frac{1}{8}{}^aA\ ^cC$, $\frac{1}{4}{}^aA\ ^dD$, $\frac{1}{4}{}^bB\ ^cC$, $\frac{1}{8}{}^bB\ ^dD$.

which is the same as the constitution of the conventional double-cross. This holds for all

(*Continued on Page* 116)

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ALBINO-DWARFISM IN HEREFORD CATTLE

A Sublethal Character

E. S. E. Hafez, C. C. O'Mary and M. E. Ensminger*

ALBINOS physiologically are animals in which pigmentation is inhibited as a result of a recessive gene. When albinos are mated with colored animals they produce young colored according to the color genes which the albinos have in their chromosomes. Completed albinism is not known for certainty in any of the farm mammals except the rabbit. However, grades of color dilution, comparable genetically with certain stages of the albino series of multiple allelomorphs in rabbits are observed in the horse[25] and the water buffalo[11, 16].

Cases of albinism in cattle have been reported in the Brown Swiss breed[11] and in other dairy breeds[5, 6, 15, 21]. A "ghost pattern" has been described in Holsteins as a faint trace of color appearing in areas where pigment normally appeared in non-albinistic cattle[5, 21]. One case of albinism in Herefords has been reported[21].

Since the work of Seligman[22] on cretinism in calves, dwarfism has been reported in both beef and dairy breeds in several countries. Hereditary dwarfism has been ascribed to a variety of genes resulting in varying anatomical anomalies such as "Bulldog"[2, 4, 24], "duckleg"[17], achrondroplasia[3, 12, 18], stumpy[1], hydrocephalus[8], akroteriasis congentia "amputated"[26], short spine[19], and "Dexter monster"[7]. Most of these defects are extremely severe in the homozygous recessive and the animals either die before birth or shortly thereafter. The Hereford dwarf has probably received more attention recently because it occurs often. Calves from dwarf × dwarf matings are phenotypically similar to dwarfs produced by heterozygous parents[20]. The phenotype of hereditary dwarfism has been described[10, 13]. It has been suggested that more than one type of dwarfism is involved. This paper reports albinism in dwarf and normal pure-bred calves.

Materials and Methods

All of the albino calves used in this study were born during the fall of 1957 in a small herd of pure-bred Hereford cattle owned by Mr. J. Shepherd near Waitsburg, Washington. The complete calving records which were available made possible a careful study of the pedigree of the albino animals. One of the albinos was a female dwarf which was brought to Washington State College for observation and measurement. The foundation cows and sire, as well as their living progeny, are assembled at the Main Experiment Station, Pullman, Washington.

Body, head and skin thickness measurements were made on the albino-dwarf and on three colored dwarfs of an age and live weight similar to the albino-dwarf. Post-mortem observations were also recorded.

Morphological Aspects

Three albino Hereford calves showed complete absence of pigmentation in the skin, muzzle and hoofs. Two albino calves were apparently normal in size; the third was an albino dwarf. The eyes of the albino dwarf, however, were not pink, owing to a slight greyish coloration of the cortex. The albino-dwarf was characterized by the protrusion of tongue and eyes accompanied by a glassy stare. It showed apparent muscular weakness

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ALBINO AND COLORED HALF-SISTER DWARFS

Figure 5

These females are from a registered Hereford herd sired by a nine-months-old purebred Hereford bull Prince Larry S. Regal. The albino dwarf, on the left, was 94 days old and the colored dwarf, on the right was 101 days of age. Both possess typical characteristics of shorter dwarf segregates that occur in the dwarf progeny of registered and commercial Herefords.

which resulted in incoordinated locomotion. This incoordination, with lateral staggering that suggested faulty equilibrium, was most apparent when the animal was induced to move suddenly or rapidly. The legs were proportionally short. The forehead and head were bulging and wide in relation to their length, and the lower jaw was undershot. An extra rudimentary incisor tooth bud was present on both sides of the regular incisor teeth.

One of the half-sisters of the albino dwarf was a dwarf of normal color pattern (Figures 5 and 6). The two normal sized albinos died at 2 and 60 days of age. The albino dwarf began to have extended paunches at the age of 12 weeks and died at the age of 14 weeks. The carrier (heterozygous) bull and cows appeared normal in size, conformation and color pattern.

Genetic Evaluation

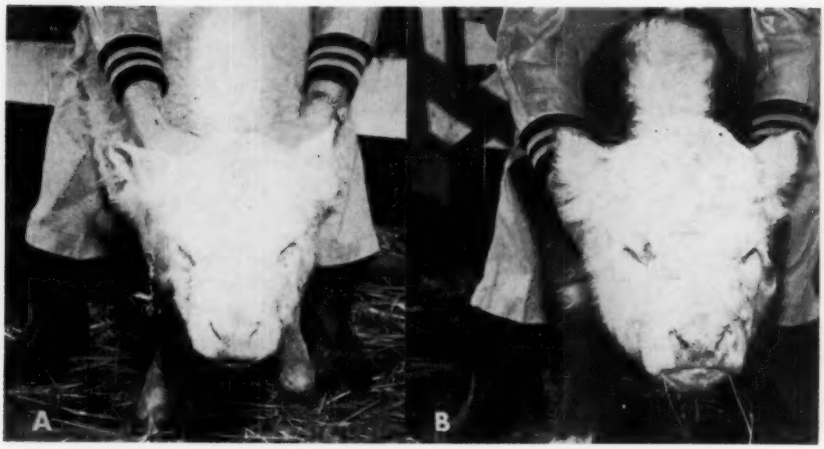
Sire "Prince Larry S. Regal", No. IV in the peligree chart (Figure 7), was mated to six different dams and produced six offspring—two albino normal-sized, one albino dwarf, one colored dwarf and two colored normal-sized calves (Table I). This appears to be a ratio of 2:1:1:2; probably involving the transmission of two genetic characters. Sire No. IV is proven heterozygous for albinism and dwarfism. At present, it is not known whether the carrier sire for number IV was number I or number II, which were not of common ancestors.

Two dams were full-sisters to the sire and produced normal-sized albinos; the

TABLE I. Offspring of purebred Hereford sire "Prince Larry S. Regal"*

Dams		Offspring	
No.†	Name	Color	Survival
6A	Princess S. Regality	Albino, dwarf	Died 14 wks.
9	Miss Larry S. Don	Albino, normal-sized	Died after birth
10	Princess S. Don	Albino, normal-sized	Died after birth
13	Miss Larry S. Baker	Colored, dwarf	Living
6	Princess S. Baker	Colored, normal-sized	Living
8A	Princess S. Mark	Colored, normal-sized	Died at birth

*Sire No. IV.
†See Figure 4.



HEAD ABNORMALITIES IN AN ALBINO DWARF AND A COLORED DWARF

Figure 6

Both the albino dwarf (left) and normal-colored dwarf (right) show the same characteristics of bulging forehead and head.

coefficient of inbreeding was 25 percent. Two dams were half-sisters from common ancestors and produced an albino dwarf and a colored normal-sized animal; the coefficient of inbreeding was 12.5 percent.

Four calves died either at birth or shortly thereafter; the mortality rate was 67 percent.

Dam No. 9 was mated twice to different sires. When mated to a non-related sire, she produced a colored non-dwarf; this is not indicated in the pedigree chart. In the second mating (to Sire No. IV or the same common ancestor) she produced an albino-non-dwarf, No. 15 in the pedigree chart.

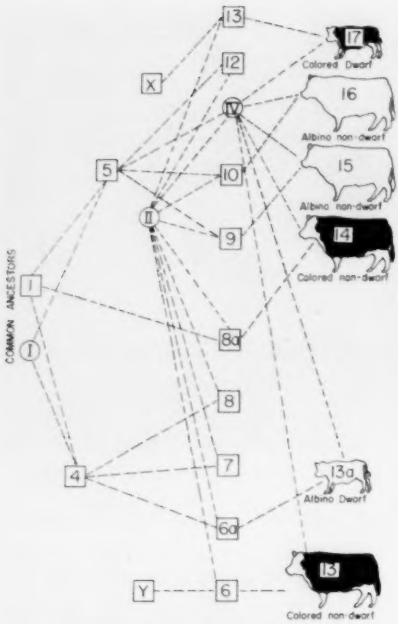
Anatomical Characteristics

Colored dwarf calves of the exact live weight and age as the albino dwarf were not available but three dwarf calves varying from 124 to 190 lbs. in weight were selected and

TABLE II. Anatomical functions of albino dwarf and colored dwarf as judged by dimension ratios

Anatomical function	Definition	Colored dwarf*		Albino dwarf†
		Range	Mean	
Live weight	lbs.	124 - 190	160	141
Ht. at withers	cm.	61.5 - 72.7	67.6	60.3
	A ratio of:			
Length index	Loin rump : body length	1:1.79 - 1:1.89	1:1.84	1:1.82
Loin-Rump index	Loin length : rump length	1:1.62 - 1:1.84	1:1.71	1:1.46
Height index	Chest depth : height	1:2.03 - 1:2.11	1:2.06	1:2.00
Width index	Hips width : width at front	1:1.18 - 1:1.48	1:1.36	1:1.00
Head index I	Width : length	1:1.36 - 1:1.65	1:1.47	1:1.51
Head index II	Face : head length	1:1.81 - 1:2.16	1:1.98	1:2.09
Head index III	Muzzle width : head width	1:1.39 - 1:1.67	1:1.53	1:1.60
Limb index	Height at rear flank : foreleg length	1:1.15 - 1:1.24	1:1.20	1:1.05
Cannon index	Length : circumference	1:1.06 - 1:1.27	1:1.16	1:1.44
Skin thickness	Average of 6 regions (mm.)	8.1 - 9.9	9.2	7.4
Dewlap depth	Average of 3 measurements (mm.)	8.5 - 11.5	10.3	6.7

*Nos. 321, 337, and 340.
†No. 13a.



RELATIONSHIP OF AFFLICTED CALVES
Figure 7

Pedigree network of six calves, Nos. 13, 13a, 14, 15, 16 and 17 from one herd. Cows are indicated by numbers in squares and bulls by Roman numerals in circles. Bull No. IV is proven heterozygous for albinism and dwarfism. One or both of the other bulls (No. I and II) are probably heterozygous for these characteristics.

used for comparison with the albino dwarf. Comparisons between anatomical functions of both types of calves were made by expressing head, limbs and body measurements in terms of ratios, keeping in mind to relate dimensions of a similar anatomical nature. The following indices were used to indicate body conformation and other anatomical functions: head, length of body, height of body, width of body, hip, limb, cannon, skin, dewlap (Table II).

The albino dwarf was similar to the colored dwarfs in the length, height and head indices. However, when compared with normal-colored dwarfs, the albino was narrow at the front in relation to width of hips, was shorter of front limb in relation to height at rear flank, and had a greater circumference of cannon in relation to length of cannon. The skin was not as thick nor the dewlap as long.

Post-mortem examination of the albino dwarf revealed no pigmentation in the walls of the rumen. There were major ulcers in both the rumen and abomasum, with deep areas of necrosis of the rumen and liver. The lungs were pneumonic with large areas of coagulation necrosis; the architecture of tissues was preserved. The thyroid was enlarged, owing to mild hyperplasia. The heart of the albino dwarf was almost spheroid, similar to that of the normal-colored dwarfs (Figure 8). An abnormal heart shape has also been reported in colored dwarf calves[9].

Post-mortem examination of the two albino normal-sized calves was not possible.

Discussion

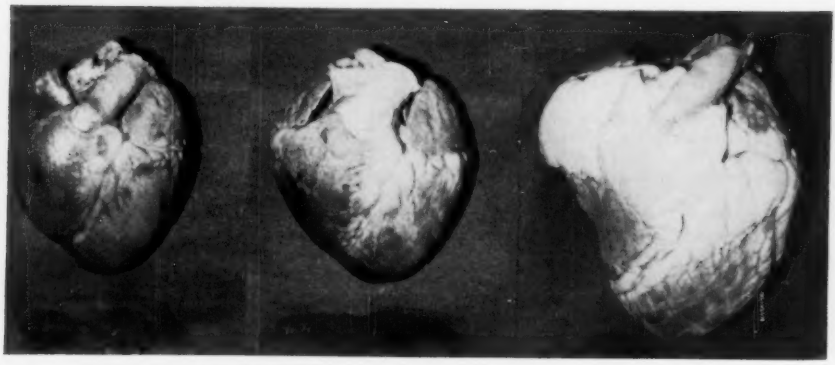
In general, albino-dwarfism appeared to be a normal or physiological dwarfism in which the body dimensions are markedly reduced as in colored Hereford dwarfs. This type of dwarf had a short, broad head and a bulging forehead associated with prognathia. The head proportion measurements were identical in both the albino and colored dwarfs, as judged by the head indices. The indices of body measurements, however, were not similar in both types of dwarfs. It has been believed that dwarfism is expressed in different patterns. Dwarfism associated with albinism may be in a specific category.

A point of great interest would be to determine whether the albino calves would acquire any body coloration if they were to survive This might very well be an albinotic dilution of color comparable to that reported by Cole *et al.*[5]

In such a trait as albino-dwarfism, the gene cannot produce the trait without the cooperation of many other genes. A gene is one factor in a very complex chemical equation. Just as the introduction of the same compound into different chemical equations does not necessarily lead to the same modification of the result, so the effect of given genes may differ according to the genetic equation into which they are introduced.

The albinism factor is possibly a subletha character. Post-mortem characteristics of the albino dwarf may or may not be associated with the expression of albinism. The high mortality rate of the offspring may be the result of specific effects associated with albinism. The death of calf number 14 might be due to calving trouble because the dam "8a" also died at calving. The lethality of factors is generally accepted as an expression of their effect in a particular environment. The dependence of the effect of lethal factors upon the external and internal environment is a feature of the action of genes in general. None of these genes produce their effects *in vacua* but all of them are highly dependent for their expression on the rest of the total genotype as well as on the environmental conditions in which they find themselves.

Physio-genetical approaches are attempted on the progeny of several mating systems, namely, a) mating to heterozygous females, b) matings to daughters of heterozygotes, c)



HEARTS OF AFFLICTED AND NORMAL CALVES

Figure 8

From left to right are shown hearts from albino-dwarf, colored dwarf, and colored control calves.

matings to full sisters, d) matings to paternal half-sisters, e) matings to daughters and f) matings to recessive types. It is conceivable that the albino breeding stock might be used to establish the genetic relationships of albinism to dwarfism. Further work will be needed before the mode of inheritance of albinism in Herefords can be analyzed. Although the two types of abnormalities, albinism and dwarfism have appeared together, the two syndromes may be independent.

Summary

Albinism and albino-dwarfism have been observed in purebred Hereford calves. The albino dwarf showed morphological and anatomical anomalies in the head similar to those of colored dwarfs. Ratios between body measurements were not identical in albino dwarfs and colored dwarfs. The albino dwarf had a basketball-shaped heart. Albinos survived from one to 94 days. The carrier animals were of normal size, conformation and color pattern. The mode of inheritance and the lethal action of albinism in Herefords is still obscure. The two syndromes, albinism and dwarfism, may be independent.

Literature Cited

1. Baker, M. L., C. T. Blunn and M. Oloufa. Stumpy, a recessive achondroplasia in Shorthorn cattle. *Jour. Hered.* 41:243-245. 1950.

2. Becker, R. B., and P. T. Dix Arnold. "Bulldog head" cattle. Prognathism in grade Jersey strain. *Jour. Hered.* 40:282-286. 1949.

3. Berger, J., and J. R. M. Innes. "Bulldog" in calves (chondrodystrophy, achondroplasia) in a Friesian herd. *Vet. Rec.* 60: 57-58. 1948.

4. Brandt, G. W. Achondroplasia in calves. *Jour. Hered.* 32:183-186. 1941.

5. Cole, L. J., E. E. Van Loon and I. Johannson. Albinotic dilution of color in cattle. *Jour. Hered.* 25:145-156. 1934.

6. Detlefsen, J. A. A herd of albino cattle. *Jour. Hered.* 11:378-379. 1920.

7. Downs, W. G., Jr. An American "Dexter monster". *Anat. Rec.* 37:365-372. 1928.

8. Ely, F., F. E. Hull and H. B. Morrison. Agnathia, a new bovine lethal. *Jour. Hered.* 30:104-108. 1939.

9. Eveleth, D. F., F. M. Bolin and M. L. Buchanan. Abnormal shape of the heart of dwarf cattle. *Vet. Med.* 51:495. 1956.

10. Gregory, P. W., W. C. Rollins, P. S. Pattengale and F. D. Carroll. Phenotypic expression of homozygous dwarfism in beef cattle. *Jour. Animal Sci.* 10:922-933. 1951.

11. Hafez, E. S. E. Unpublished data, Cairo University. 1957.

12. Johannson, I. A new type of achondroplasia in cattle. *Hereditas* 39:75-87. 1953.

13. Johnson, L. E., G. S. Harshfield and W. McCone. Dwarfism, an hereditary defect in cattle. *Jour. Hered.* 41:177-181. 1950.

14. Keller, K. Die Bedeutung der modernen Verebungslehre fur die praktische, namentlich die landwirtschaftliche Tierzucht. *Zuchtungskunde* 6:44-58. 1931.

15. Kroon, H. M., and G. M. v.d. Plank. Einige subletale Faktoren bei Haustieren in den Niederlanden. *Biologia Generalis* 8:213-218. 1932.

16. Levine, C. O. Albino water buffaloes. *Jour. Hered.* 16:66. 1925.

17. Lush, J. L. "Duck-legged" cattle on Texas ranches. *Jour. Hered.* 21:85-90. 1930.

18. Mead, S. W., P. W. Gregory and W. M. Regan. A recurrent mutation of dominant achondroplasia in cattle. *Jour. Hered.* 37:183-188. 1946.

19. Mohr, O. L. and C. Wriedt. Short

spine, a new recessive lethal in cattle; with a comparison of the skeletal deformities in short spine and in amputated calves. *Jour. Genet.* 22:279-298. 1930.

20. Pahnish, O. F., E. B. Stanley and C. E. Safley. A study of homozygous dwarfism in beef cattle. *Proc. West. Sec. Amer. Soc. Anim. Prod.* 1952.

21. Peterson, W. E., L. O. Gilmore, J. B. Fitch and L. M. Winters. Albinism in cattle. *Jour. Hered.* 35:135-144. 1944.

22. Seligman, C. G. Cretinism in calves. *Jour. Path. and Bact.* 9:311-322. 1904.

23. Stonaker, H. H. and R. C. Tom. "Compact" Shorthorns. *Jour. Hered.* 35: 247-250. 1944.

24. Sturrarrer, T. C. Bulldog and hairless calves. *Jour. Hered.* 34:175-178. 1943.

25. Wriedt, C. Albinisme i Hester. *Tidskr. f. d. norske Landbrug Heft* 10:396-404.. 1918.

26. Wriedt, C. and O. L. Mohr. Amputated, a recessive lethal in cattle: with a discussion on the bearing of livestock breeding. *Jour. Genet.* 20:187-215. 1928.

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A Modification of the "Double-Cross" Technique of Breeding Hybrid Poultry

(*Continued from Page* 110)

loci, in particular the number of loci heterozygous in the average individual of the population will be the same as in the conventional double-cross.

In fact apart from specific epistatic effects and possible slight recombination and maternal effects this cross is exactly the same as the double-cross.

If the F_2 animals are bred together at random and in sufficient quantity to avoid inbreeding effects, they will produce a population having an identical composition with the F_2. This can be maintained by random mating with its composition unchanged except by the effects of natural selection and recombination, owing to the Hardy-Weinberg Law, and the fact that the F_2 (but not the F_1) is a population in equilibrium.

Such "F_2 populations," which have the same yields as their parent F_2's, should have higher yields than the "mid-parent value" (the mean yield of their parent inbred lines) though not as high as their parent F_1's. No data on the comparative yields of F_2's of this type appear to be available, and so they must be found by experiment; however present knowledge suggests that they may be close to half way between the mid parent value and that of the F_1.

These F_2 populations presumably will be free from some of the inbreeding depression and the consequent low yield and hatchability characteristic of inbred lines, and may prove much more attractive commercially than inbred lines. At any rate they give a method of producing double-cross birds commercially with only three matings. The F_1's are made up, the F_2's and F_2 populations are bred in large flocks, and the two flocks cross-mated to give the "F_2 population double-cross" generation. If this proves as similar to the conventional double-cross in fact as in theory the parent lines may then be discarded. Thereafter only two flocks have to be maintained and the cross between them made, thus reducing the trouble and expense of breeding a double-cross to no more than that of breeding a cross between two normal breeds.

If a double-cross should be made with parents derived from two different breeds the F_1's (and hence the F_2 populations) probably should be made from two inbred lines from the same breed, and only the final F_2 population double-cross should be a breed cross.

Literature Cited

1. Pease, M. Inbreeding in poultry livestock improvement. Official report of the Eighth World Poultry Congress. 1948.

2. ———— and F. Dudley. Hybrid vigour in poultry. Northern Poultry Breeding Station, Reaseheath, Cheshire, England. Section papers of the Tenth World Poultry Congress. 1954.

SCURS IN A HERD OF ABERDEEN-ANGUS CATTLE

R. L. Blackwell and J. H. Knox*

An autosomal gene in cattle, *Sc*, which makes otherwise polled cattle have scurs, is considered to be sex-influenced to the extent that it is dominant in males and recessive in females[1]. Thus a bull with scurs would carry at least one *Sc* gene. In a breeding herd of Aberdeen-Angus cattle where the males and females had no scurs and the frequency of the *Sc* gene was low but not zero, only females would carry the gene in the heterozygous state. Under these circumstances, the proportion of heterozygous females in the cow herd would be twice the frequency of the *Sc* gene. The gene frequency in the herd bulls would be zero. Since females with scurs must be homozygous (*Sc Sc*) and would result only from matings in which both parents transmitted the gene, no scurred female offspring would be expected. Male offspring would be expected to appear with the same frequency as that of the *Sc* gene in the cow herd.

If a scurred bull were used in such a cow herd as that just described, approximately half his male offspring should have scurs if the bull was heterozygous. Actually the expected frequency of scurred male offspring should slightly exceed one-half because of the few *Sc* genes transmitted by heterozygous cows to their sons which did not receive the gene from their heterozygous sire. Daughters with scurs would be expected to appear with frequency one-half that of the gene in the cow herd.

Some supporting evidence for this mode of gene action has been obtained for the *Sc* gene from a relatively large herd of Aberdeen-Angus cattle. This is reported here together with an instance in which a bull with scurs failed to sire the expected proportion of sons with scurs.

The Data

A herd of Aberdeen-Angus cattle located in the state of Chihuahua in northern Mexico has been under observation from 1944 through 1957. All bull and heifer calves produced in a select portion of the herd were observed at the age of six to eight months when records on each calf were taken. This part of the herd was managed so that pedigree information was available on each calf. Records of the occurrence of scurred and non-scurred progeny were compiled. Castration of bull calves was done only after weaning.

During the period this herd was under observation, a portion of the herd sires were produced in the herd itself, and after 1951 all sires used came from the herd. The breeding system was essentially random within this closed herd, except matings were avoided that would result in appreciable inbreeding. The breeding season was unrestricted, and calves were weaned at about three-month intervals.

One particular bull, Almirante, had been produced in the herd and was used in the latter years. He had small scurs and was assumed to be heterozygous since his sire had no scurs. He was the only bull with scurs that was used during the period studied. Calves sired by him were observed from 1954 through 1957. These matings constitute a test of the hypothesis that he was heterozygous for the *Sc* gene.

A total of 1829 bull calves were observed for the presence or absence of scurs during the 14-year period. Of these, 43 had scurs. No scurred heifer

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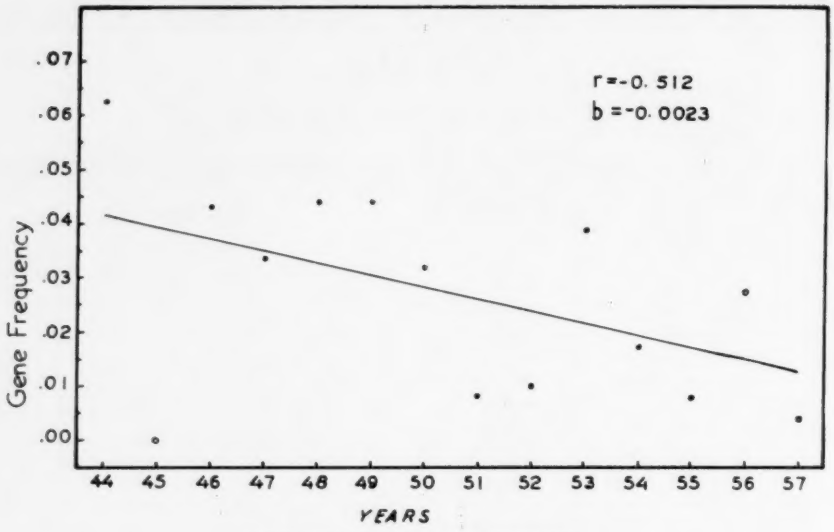

DECLINE IN GENE FREQUENCY

Figure 9

The chart above shows the change in estimated frequency of the *Sc* gene.

calves were produced in this portion of the herd. The data tabulated by years for all bull calves are presented in Table I. Table II gives information on the sons of Almirante and the sons of non-scurred bulls that were observed during the period 1954-1957.

TABLE I. The number of bull calves observed annually, the number with scurs and the percentage with scurs

Year	Number of calves	Number with scurs	Percent with scurs
1944	144	9	6.25
1945	62	0	0.00
1946	93	4	4.30
1947	60	2	3.33
1948	45	2	4.44
1949	112	5	4.46
1950	94	3	3.19
1951	120	1	0.83
1952	192	2	1.04
1953	105	4	3.81
1954	237	4	1.69
1955	128	1	0.78
1956	187	5	2.67
1957	251	1	0.40
Total	1829	43	2.35

Results and Discussion

The percentage of scurred male offspring for the entire period was 2.35 percent. During the period 1954-1957, Almirante sired the same percentage of scurred bull calves as did the non-scurred bulls (1.37 percent). This particular scurred bull apparently was not transmitting the *Sc* gene to his sons, and presumably not to his daughters, even though his phenotype indicated that he should. Since only one scurred son appeared in his progeny, the gene most likely came from the dam rather than the sire. The probability that a heterozygous bull would sire only one scurred son in 73 is extremely small. It must be concluded from the progeny test that this particular scurred bull was not heterozygous for the *Sc* gene. The genetic explanation for his having scurs but not transmitting the gene is not clear.

The proportion of scurred bulls born gives a

TABLE II. The number of bull calves observed during the period 1954-1957, the number with scurs and the percent with scurs sired by bulls with and without scurs

Phenotype of the sire	Number of calves	Number with scurs	Percent with scurs
Non-scurred	730	10	1.37
Scurred	73	1	1.37
Total	803	11	1.37

direct estimate of the frequency of the *Sc* gene in the cow herd from which they came, provided there were no *Sc* genes transmitted by their sires. This assumes also that the gene was carried only by heterozygous cows. Using all the data, including that from the sons of Almirante, estimates of the gene frequency each year were obtained. The mean of these yearly estimates gave an estimate of 0.0266 as the frequency of the *Sc* gene for the 14-year period.

There appeared to be a noticeable but erratic decline in the percentage of bull calves born with scurs each year. Such a trend, if real, would indicate a decline in the frequency of the *Sc* gene. The number of bull calves with scurs observed annually deviated significantly from the expected number ($\chi^2 = 23$, d.f. $= 12$). The expected number was based on the percentage of bull calves with scurs produced for the entire period (2.35 percent). Undoubtedly most of the year-to-year variability was due to chance transmission of the gene from dam to son, while some could be due to a change in gene frequency in the cow herd.

To determine whether the apparent decline was real, the trend in gene frequency was studied. The linear regression of the yearly estimates of gene frequency on time approached significance at the five percent level of probability ($P < .10$). There was no evidence of curvilinear regression. The estimated rate of change in gene frequency was -0.0023 ± 0.0011 per year, indicating that a slow decline may have been taking place in this herd (Figure 9). This point was studied further by comparing the average frequency for the first seven years (0.037) with that for the last seven years (0.016). The difference (0.021 ± 0.0086) was significant at the five percent level of probability. This tends to increase confidence in the regression coefficient obtained earlier. Assuming that approximately three generations in cattle are represented by this 14-year period and the gene frequency was changing at the rate of -0.0023 per year, the rate of change in gene frequency per generation was approximately -0.011. The continued use of bulls that were free of the *Sc* gene would effect a 50 percent reduction in gene frequency per generation. Thus the gene frequency would approach zero asymtotically. In the range of values determined by the regression analysis through which gene frequency would pass, 0.04 to 0.01, it would be difficult to detect a change in gene frequency much more critically than has been done here.

Summary

The frequency of the gene for scurs, *Sc*, was studied in a relatively large herd of Aberdeen-Angus cattle. An estimate of the frequency of this gene was obtained from the proportion of scurred bull calves produced in the herd. The average gene frequency during the 14-year period, 1944-1957, was 0.0266. During this period the change in gene frequency determined by a regression analysis was estimated to be -0.0023 per year, resulting in a decline from approximately 0.04 in 1944 to 0.01 in 1957. Although small, the change was considered to be real.

From a total of 73 bull calves sired by a bull with small scurs only one had scurs. It is highly improbable that this sire was heterozygous for the *Sc* gene. Except for this instance, the accepted mode of gene action for the *Sc* gene appears to be clearly in evidence in this herd.

Literature Cited

1. Shrode, Robert R. and Jay L. Lush. The genetics of cattle. *Advances in Genetics* I. 209-261. 1947.

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Closing Date for Basic Research Proposals Announced By the National Science Foundation

The Division of Biological and Medical Sciences of the National Science Foundation announces that the next closing date for receipt of basic research proposals in the life sciences is September 15, 1958. Proposals received prior to the Foundation's Advisory Panels and disposition will be made approximately four months following the closing date. Proposals received after the September 15, 1958, closing date will be reviewed following the Winter closing date of January 15, 1959.

Inquiries should be addressed to the National Science Foundation, Washington 25, D. C.

VARIEGATED LEAVES

Figure 10

The first and second leaves of variegated *pg*^{m} plants are shown above. The counting area was a uniform distance from the tip of each leaf.

THE EFFECT OF TEMPERATURE ON THE MUTATION RATE OF A MUTABLE LOCUS IN MAIZE

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THE role of temperature on the induction of mutation has long been considered an important aspect of gene mutation study. Two types of investigations can be included here: (1) temperature effect on the over-all mutation rate of a random sampling of genes and (2) the effect of temperature on mutable (highly unstable) genes.

Muller[10] and Timoféeff-Ressovsky[16] found a 2- to 3-fold increase in both visible and lethal mutations in *Drosophila* with a 6-7°C increase in temperature. Temperature shocks above and below a normal optimum applied to *Drosophila* larvae produce an increase in mutation frequency in proportion to the shock[6,14]. In *E. coli*, Witkin[18] observed a higher mutation rate when the higher temperature is associated with the first division following radiation.

In temperature experiments with mutable genes, the reverse effect has generally been found. Demerec[3], using the unstable gene, miniature 3 gamma of *Drosophila virilis*, observed no difference in mutation rate at 20°, 25° and 30°C. Rhoades[15], working with the a_1-*Dt* complex in maize, found a depressive effect with an increase in temperature: that is, the higher temperature was accompanied by a decrease in mutation rate. Fabergé and Beale[5] also reported an apparent diminution in mutation rate with increase in temperature. Similar results have been reported in other experimental studies of temperature differences with variegated genes[4,7].

The effect of temperature on the rate of mutation of *pg* (pale green) to *Pg* (green) can be readily determined due to the facility of counting the green stripes on a pale green background. In addition, an approximate determination of the post-germination development of the first, second and third leaves of the corn seedling can be made from a study of the linear lengths of the stripes. An abstract of the material in this report has been presented[12].

Materials and Methods

In the pale green mutable series, *pg*, pale green, mutates to *Pg*, green, and these mutations are recognized by the appearance of green stripes in a pale green background (Figure 10). Seedlings of this type are designated pg^m. Mutability is controlled by Enhancer, *En*, which is located at the *pg* locus, $\widehat{pgEn}$, or on a chromosome segregating independently of *pg*, *pg En*. In the absence of *En*, the *pg* locus is stable[13].

In these experiments 16 different sources of uniformly mutating pg^m seedlings, chosen for their clear expression (Figure 10), were used. The seeds from each of the 16 families were divided into two groups: one group was kept at a constant temperature of 16°C and the other maintained at 28°C. The temperature fluctuated ±2°. Both $\widehat{pgEn}$ and *pg En* types were included and no distinction is made between them in the final analyses of the data since they responded identically to the treatment.

In order to insure uniformity in germination seeds were planted in soil with the epicotyl pointing upwards. At the beginning of germination the flats of seedlings were put in a constant temperature box kept at the indicated temperature for treatment where they remained throughout the course of the experiment. This cork-jacketed box was approximately 5 × 5 × 4 ft. and located in a room in which the temperature was

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maintained at 30°C. Temperature regulation in the box was in the form of a cooling system with a constant air blower for continuous circulation. Six 46 in. fluorescent tubes were used as the source of artificial light so that all the seedlings would be equally exposed. No attempt was made to maintain a constant humidity, but soil moisture kept it high.

Mutation rate is calculated from the number of individual stripes in a leaf and the length of each stripe is a function of the number of cell divisions following an initial mutation.

Counts of the green stripes were made under a dissecting microscope on the first three seedling leaves at the same stage of development in both the 28°C and 16°C experiments. The length of the third leaf blade (13-15 cm) served as the criterion for comparison. A standard area, 6.5 × 15 mm, located 2, 13 and 19 mm from the tip of the 1st, 2nd and 3rd leaf, respectively, was used to make the counts in both treatments. Size of the individual stripes was determined by comparing them with millimeter ruled paper. This makes the hundredth millimeter measurements only approximate. Since the length of the bundle sheath cells averages the same in both treatments (.048 to .051 mm), it can be assumed that the same number of cells are included in the area counted. Corrections were made for large wide stripes resulting from early occurring mutations that would otherwise have masked mutations resulting from the treatment.

In experimental families Te 9-16, the cold and warm treatments were given at different times in the same box, while experiments Te 1-8 were run simultaneously in two different boxes.

Counts were made at different intervals after planting; 9 to 14 days in the 28°C experiments, and 24 to 44 days for families in the 16°C experiments.

TABLE I. Temperature and family differences in the mutation rate of pg^m. Analysis of variance (Anderson and Bancroft,[1] p. 278)*

Source of variation	df	Mean square	Actual F ratio	F value 5%	F value 1%
Temperature	1	8,136**	>800	3.86	6.70
Family	9	24.7**	2.50	1.90	2.46
T × F	9	43.57**	4.41	1.90	2.46
Error	397	9.88			

*The author is grateful to Mr. James Smith for the analysis of variance computations.

Results

(a) Effect of temperature on mutation rate

A strikingly higher mutation rate is found among the seedlings developing at 28°C than among the seedlings grown at 16°C (Table I). This higher rate was found in all families and in each leaf. The difference between the treatments is highly significant. The difference between the families is also highly significant.

(b) Difference in increase in mutation rate among the three seedling leaves

In comparisons within families and between the first, second and third leaves under the two treatments, a greater increase in mutation rate is evident in the younger leaves. The increase in mutation rate in the third (younger) leaf (12.8×) is greater than in the first (older) (5×) or second (8.2×) and greater in the second than in the first leaf (Table II).

(c) Importance of length of stripe among the different leaves in the 28°C treatment

From this temperature experiment, it is also possible to estimate the approximate number of cells that arise in each of the first three leaves from the time of germination until the time of counting. It was observed while counting the stripes in the leaves in the 28°C series that the majority of the stripes in each leaf group were within a specific size range: the majority of stripes in the first leaf were .5 mm

TABLE II. Differences in increase in mutation rate between the three leaves

Leaf	28°C Families: Total number of stripes (73 plants)	28°C Families: Mean number of stripes per leaf	16°C Families: Total number of stripes (66 plants)	16°C Families: Mean number of stripes per leaf	Ratio 28°C/16°C
1	3981	54.5	663	10.0	5.45
2	4220	57.8	463	7.0	8.25
3	2998	41.1	212	3.2	12.8

long or less while in the second leaf they were 1 mm or less and in the third, 1.5 mm or less. (Data concerning temperature effect on the rate of mutation, as discussed above, were derived from a comparison of the number of these stripes at different temperatures). This is evident in Table III, as is also the fact that stripes longer than those just described are approximately equal in number in both the cold and the warm series. These longer stripes are assumed to have originated prior to the treatment and would therefore be expected to be distributed equally among the leaves of seedlings at both temperatures. Therefore each shorter stripe is in effect the result of mutation following the initiation of the temperature treatment.

The appearance in the 16°C treatment of some stripes in leaf 1 less than .5 mm, in leaf 2 less than 1 mm, and in leaf 3 less than 1.5 mm indicates that mutations can occur at 16°C but do so less frequently.

(d) Cell division as related to the length of stripes in developing leaves of germinating seedlings

The mean linear length of chlorenchyma cells of the second maize leaf in the treated strains is approximately .032 mm in both the cold and warm treatments. Since the longest stripes resulting from the treatment are .5 mm in leaf 1, 1 mm in leaf 2 and 1.5 mm in leaf 3 (Table III), then these stripes would contain approximately 15, 31, and 47 cells, respectively. How many divisions would be necessary to account for these cells cannot be determined from this experiment.

Discussion

In contrast to previous findings on the effect of temperature on mutable genes, the mutable pale green allele (pg^m) shows a decided increase in mutation rate with an increase in temperature. This difference can be reconciled, if, as suggested by McElroy and Swanson, a cell contains competing mutagen and inhibitor systems—either one of which may be preferentially affected by an increase in temperature. (These systems are considered to be competing for a common limited substrate.) If the increase in temperature favors an inhibitor system rather than a mutagen system, the higher temperature would lead to a decrease in mutation rate. Under this hypothesis, in the case of pg^m, increased temperature would favor a mutagenic substance. This mutagenic substance could thus be assumed to be produced by *En*.

Previous reference to the nature of the gene based on the effect of temperature[9,17] includes the theory of energy states in order to account for the varied rates of gene mutation that were observed. But McClintock[8] and Brink[2] have presented evidence that the mutation event involving changes of mutable genes represents the loss or removal of some particulate element closely allied to the unstable gene. According to present ideas involving mutable genes, some element such as *Ac*, *Mp* or *En* either at the locus or assorting independently of the locus in question accelerates the removal of the inhibiting material, thus exposing the locus. (In the case of *En*, warm temperatures would accelerate this removal.) Under this hypothesis the application of the theory of variable energy levels associated with the varied rates of mutation in mutable genes is not valid since the gene molecule itself remains unchanged.

Time factor in relation to mutation

Since the 16° plants took 2 to 3 times as long to develop to the same stage as did the seedlings grown at 28°C, there would be a greater opportunity for mutations to occur if mutation rate were a function of time. If mutations in pg^m arise from errors in copying (i.e. faulty gene reproduction), a proportionate increase in mutation rate might result if the duration of the process of cell division is increased. That is, there might be a greater chance for error over a longer period than over a shorter period. On the basis of this hypothesis, one might expect a higher mutation rate in the 16°C group. (Of course, this assumes that an "error" can occur at any point in gene multiplication.) Obviously this is not so since fewer mutations occurred at the lower temperature. The temperature study on pg^m shows mutation rate to be independent of time. This is in agreement with the findings of previous investigators[11].

In comparing the mutation rate among the three leaves (Table II), it is evident that the younger leaves (third and second) show a greater increase in mutation rate than does the first. It is assumed that these three leaves differ in the number of cell divisions which occur during the course of the treatment. On the basis of a higher rate in the younger leaves, it appears that the greater the number of dividing cells subjected to the treatment, the greater is the chance for mutation.

Cell divisions occurring in seedling leaf growth

The occurrence of cell divisions in the growing leaves during the germination process is

TABLE III. Total number of different sized stripes in the first three leaves of two treated families

		Family 26 (9 seedlings)		Family 34 (18 seedlings)	
	Length of stripe	28°	16°	28°	16°
Leaf 1	.4 mm and less	293	4	587	5
	.5 mm and less	353	19	766	8
	1 mm and less	355	27	770	8
	1.1 mm and above	16	35	93	101
2	.5 mm and less	188	11	610	15
	.8 mm and less	412	12	635	15
	1 mm and less	485	36	858	50
	1.1 mm and above	13	27	40	47
3	.5 mm and less	45	0	114	8
	.8 mm and less	84	0	153	8
	1 mm and less	200	1	571	29
	1.5 mm and less	246	1	572	36
	1.6 mm and above	24	23	82	76

confirmed by the difference in mutation rate (number of mutant stripes) found between the 28° and 16° treatments. If cell divisions do not occur, then all the cells are *a priori* deposited during the embryo stage. Under these circumstances, with all the cells already formed, no difference would be expected between the two treatments. In addition, the appearance of longer stripes in the younger leaves suggests that these leaves undergo more divisions than the older leaves during the germination process.

These data on cell division derived from the study of mutant stripes imply a random occurrence of cell origins in the maize leaf blade. The stripes appear to have no relation to a basal or marginal meristem.

Summary

The mutation rate of the unstable gene, *pg*^{m}, is higher at 28° C than at 16°C.

Mutations can occur at 16°C but occur less frequently.

A greater increase in mutation rate is observed in the younger leaves (12.8×), suggesting that the more cell divisions, the greater the opportunity for mutation to occur. In the first leaf and second leaf the increase is 5.4× and 8.2× respectively.

Cell divisions are postulated to occur in the leaf blade during the germination process and more occur in the younger leaves than in the older ones. It is estimated that 15, 31 and 47 new cells arise between the time of the initial stage of germination and time of counting in the first, second and third leaf respectively.

The mutation process of mutable genes is considered not to involve changes in the energy level of the gene.

Literature Cited

1. Anderson, R. L. and T. A. Bancroft. Statistical Theory in Research. McGraw-Hill Book Company. New York. 399 pp. 1952.

2. Brink, R. A. and R. A. Nilan. The relation between light variegated and medium variegated pericarp in maize. *Genetics* 37:519-544. 1952.

3. Demerec, M. Effect of temperature on rate of change of the unstable miniature-3 gamma gene of *Drosophila virilis*. *Proc. Nat. Acad. Sci.* 18:430-434. 1932.

4. Eyster, W. H. The effect of environment on variegation patterns in maize pericarp. *Genetics* 11:372-386. 1926.

5. Fabergé, A. C. and G. H. Beale. An unstable gene in *Portulaca:* Mutation rate at different temperatures. *Jour. Genet.* 43:173-187. 1942.

6. Goldschmidt, R. B. Experimentelle Mutation und das Problem der sogenannten Parallelinduktion. Versuche an *Drosophila*. *Biol. Zbl.* 49:437-448. 1929.

7. Gowen, J. W. and E. H. Gay. Chromosome constitution and behavior in eversporting and mottling in *Drosophila melanogaster*. *Genetics* 19:189-208. 1934.

8. McClintock, Barbara. Chromosome organization and genic expression. *Cold Spring Harbor Symp. Quant. Biol.* 16:13-17. 1951.

9. McElroy, W. D. and C. P. Swanson. The theory of rate processes and gene mutation. *Quart. Rev. Biol.* 26:348-363. 1951.

10. Muller, H. J. The measurement of gene mutation rate in *Drosophila*, its high variability, and its dependence on temperature. *Genetics* 13:279-357. 1928.

11. Novick, Aaron and Leo Szilard. Experiments on spontaneous and chemically induced mutations of bacteria growing in the chemostat. *Cold Spring Harbor Symp. Quant. Biol.* 16:337-343. 1951.

12. Peterson, Peter A. Effect of temperature on the mutation rate of a mutable locus in maize. (Abstract). *Genetics* 37:614. 1952.

13. ————. A mutable pale green locus in maize. *Rec. Genetics Soc. Am.* 22:92-93. 1953.

14. Plough, H. H. and P. T. Ives. Induction of mutations by high temperature in *Drosophila*. *Genetics* 20:42-70. 1935.

15. Rhoades, M. M. The genetic control of mutability in maize. *Cold Spring Harbor Symp. Quant. Biol.* 9:138-144. 1941.

16. Timoféeff-Ressovsky, N. W. The experimental production of mutations. *Biol. Rev.* 9:411-457. 1934.

17. ————, K. G. Zimmer and M. Delbruck. Über die Natur der Genmutation und der Genstruktur. *Nachr. Ges. Wiss. Gottingen, N. F.* 1:189-245. 1935.

18. Witkin, E. M. Effect of temperature on spontaneous and induced mutation in *E. coli*. *Proc. Natl. Acad. Sci.* 39:427-433. 1953.

THE OCCURRENCE OF CONGENITAL PORPHYRIA IN HOLSTEIN-FRIESIAN CATTLE

D. E. Madden, D. J. Ellis, R. D. Barner, I. Melcer and J. M. Orten*

FOR over 20 years the literature has contained occasional reports on the occurrence in cattle of a rare metabolic disorder known as congenital porphyria (also referred to as congenital porphyrinuria, pink tooth, osteohaemochromatosis, and hematoporphyrinuria). Cattle affected with this condition exhibit a syndrome of effects manifested early in life. The usual symptoms are discolored (various shades of pink to reddish-brown) teeth and urine. Dermal lesions on unpigmented areas may be present when the animals are subjected to sunlight. Anemia and unthriftiness also may be observed. Discolored bones are observed upon post-mortem examination.

Review of the Literature

While earlier literature reports are available on human and animal cases of porphyria, the first animal cases to be described clinically were 13 grade Shorthorn cattle (10 males and three females) noted by Fourie[6]. These cattle were sired by the same normal appearing bull, which suggested the hereditary transmission of the condition. Fourie[7] reported that bovine congenital porphyria is inherited as a recessive character. This conclusion was based on evidence obtained when he mated this carrier Shorthorn bull to: (a) 10 unrelated females and obtained eight normal calves; (b) two cows, each of which previously had produced an affected calf, and obtained one normal calf; (c) three of his normal appearing daughters and obtained three normal calves, one affected female and two normal calves, and one affected male and two normal calves, respectively; (d) an affected daughter which produced an affected calf. Fourie[7] cited two cases of affected Friesland heifers resulting from mating a Friesland bull with his own normal appearing daughters. This bull was related to a previously described affected grade Friesland cow[8].

Other cases of bovine congenital porphyria have been reported in several breeds in various countries, including a yearling Hereford steer in Arkansas[2] and one in Tennessee[13], three cases in Canada with the breed not stated[14], several Shorthorn cases in Denmark[10], four Shorthorn females in Britain[1,16], two Holstein heifer calves in California[15], and three Holsteins in New York[4]. Jorgensen and With[11] have verified 18 affected males and 12 affected females (Shorthorn and Danish Black and White milk cattle). These latter workers indicated the condition to be inherited as a recessive.

Congenital porphyria appears to be inherited as a dominant in swine and several workers have observed such cases[3,5,9,10].

Results and Discussion

The recent occurrence in Michigan of 11 purebred Holstein-Friesian cattle exhibiting the symptoms of congenital porphyria prompted the authors to consider the genetic relations of these animals. The genetic relations, clinical descriptions, and urine studies are presented below. The mating of affected animals is being attempted.

Genetic relations

The genetic relations of 11 purebred Holstein-Friesian animals from five different

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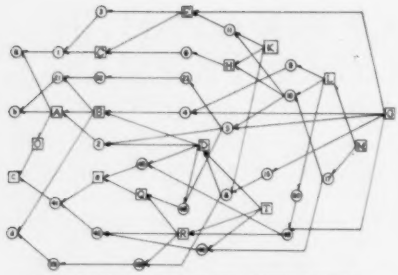
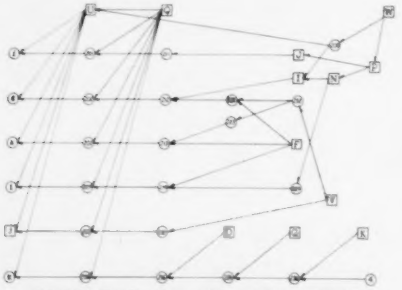


PEDIGREE OF FIVE AFFLICTED ANIMALS

Figure 11

Pedigrees of five bovines affected with congenital porphyria in Herds 1 to 4. Squares represent males and circles represent females.



PEDIGREE OF SIX AFFLICTED ANIMALS

Figure 12

Pedigrees of six bovines affected with congenital porphyria in Herd 5.

herds are presented in Figures 11 and 12. Lower case letters indicate affected animals, while capital letters or numerals designate phenotypically normal animals. Numerous normal animals of similar breeding also are present in these herds.

Figure 11 indicates that male *A* has sired two daughters and a grandson with the condition and is a paternal half-brother to affected female *d*. Since male *B* has sired affected female *d*, one may conclude that males *A* and *B* and females *1*, *21*, *41*, and *101* are carriers. The presence of affected male *e* indicates that male *Q* and female *45* are carriers. Because of the complex interrelations farther back in the pedigrees, it is difficult to say accurately from where the carrier animals received the gene, although the parents of each affected animal trace to males *G*, *K*, *L*, and *M*.

The relations noted in Figure 11 indicated that male *Q* is a carrier. Consequently a herd was visited which was known to be following a breeding program aimed at increasing the relationships to male *Q*. According to the owner, this herd (Herd 5) had not, prior to examination, contained any animals affected with congenital porphyria. However, upon examination of the teeth of young animals in the herd (approximately 40 head under one year of age), six animals with discolored teeth were observed. These results are presented in Figure 12 to indicate the situation in Herd 5 and to demonstrate the relations with those presented in Figure 11. As noted in Figure 12, the parents of the five affected heifers and one affected male are offspring of male *Q* and are therefore related at least as closely as half-sibs.

The parents of affected animals *a* through *d* and *f* through *k* have been observed and appear normal. Each dam has produced at least 400 pounds of butterfat in a lactation and the sires have many offspring. Affected and normal offspring from normal parents lends support to previous reports that the condition is inherited as a simple recessive[7]. The normal production and fertility of the carrier animals suggests that the gene in the heterozygous condition has no detrimental effects.

Carrier females *1*, *21*, *41*, and *201* to *206* are currently in production in two cooperating herds. Carrier males *A*, *Q*, and *U* are in active service. Two affected animals (female *b* and male *e*) and others as they become available, are being mated in order to definitely establish the simple recessive nature of the condition. Such critical matings have not been reported to date in the literature.

Clinical descriptions of affected animals

Animal *a* (female) was born in November 1955 in Herd 1 and died of pneumonia at ten months of age. Discolored teeth and bones were observed upon post-mortem examination.

Animal *b* (female) was born in September 1956 in Herd 1 and currently is at the University. This heifer appears normal except both urine and teeth are discolored reddish brown. The heifer was on pasture all summer but showed no evidence of photosensitization.

Animal *c* (male) was born in November 1956 in Herd 1 and died at three days of age. Discolored teeth and bones were observed upon post-mortem examination.

Animal *d* (female) was a producing cow, born in Herd 2 in July 1953 and slaughtered in 1956. This cow produced over 10,000 pounds of milk in her first lactation in Herd 3. Sloughing of udder and teat tissue similar to sunburn was observed and required several months to heal. Discolored bones were observed when she was slaughtered but no report is available concerning her teeth.

Animal *e* (male) is a fertile five-year-old male born in Herd 2 and used for a short time in Herd 1 and for an extended period in Herd 4. This bull, recently brought to the University, is anemic and unthrifty. Lesions of

photosensitization are evident on the unpigmented areas of the back and scrotum. The teeth are discolored, although not as dark as the teeth of the heifer *b*. The urine is dark reddish brown. Cow *45*, the dam of male *e*, is said to have had dermatitis but this condition apparently did not affect her production. She produced an average of over 12,000 pounds of milk for four lactations.

Animals *j* (male) and *f*, *g*, *h*, *i*, and *k* (females) in Herd 5 when observed ranged from one to 10 months of age. Discolored teeth and urine were present but no areas of photosensitization were observed.

As noted above, discolored urine was observed in several animals. Normal appearing urine has been observed also from affected animals. This urine usually becomes darker upon standing or on exposure to sunlight. The normal appearing urine from affected animals contains abnormal amounts of porphyrins.

Urine studies

Using both paper and silica gel chromatographic techniques, urine samples from animals *b*, *e*, *f*, *g*, *h*, *i*, *j*, and *k* were found to contain relatively large amounts of porphyrins and of porphobilinogen. The porphyrins were predominantly uroporphyrin type III and coproporphyrin type III with smaller amounts of coproporphyrin type I and other porphyrins, possibly including protoporhyrin. The total porphyrin excretion per 24 hours was in the range of 50 to 100 mg. This finding is in general agreement with those of Jorgensen and With[10] in studies of porphyria in cattle in Denmark, and Amoroso, *et al.*[1] in England. Details of the present chemical studies are being reported elsewhere[12].

Summary

The genetic relations and clinical aspects of 11 purebred Holstein-Friesian cattle with congenital porphyria are discussed. The evidence reported seems to substantiate previous reports that congenital porphyria in cattle is inherited as a simple recessive.

Literature Cited

1. Amoroso, E. C., R. M. Loosmore, C. Rimington and B. Tooth. Congenital porphyria in bovines: First living cases in Britain. *Nature* 180:230-231. 1957.

2. Anonymous. Porphyrinuria in a yearling Hereford. *Vet. Med.* 34:102-103. 1939.

3. Barner, R. D. Unpublished data. 1956.

4. Bentinck-Smith, John. Personal communication. 1957.

5. Clare, N. T. and E. H. Stephens. Congenital porphyria in pigs. *Nature* 153:252-253. 1944.

6. Fourie, P. J. J. The occurrence of congenital porphyrinuria (pink tooth) in cattle in South Africa. *Onderstepoort Jour. Vet. Sci.* 7:535-566. 1936.

7. ————. Bovine congenital porphyrinuria (pink tooth) inherited as a recessive character. *Onderstepoort Jour. Vet. Sci.* 13: 383-398. 1939.

8. ———— and C. Rimington. A further case of congenital porphyrinuria (pink tooth) in a living grade Friesland cow in South Africa (Cedara case). *Onderstepoort Jour. Vet Sci.* 10:431-436. 1938.

9. Jarmai, K. Uber die osteohamochromatosis des schweines. (Abst.) *Vet. Bull.* 12:459. 1942.

10. Jorgensen, S. K. and T. K. With. Congenital porphyria in swine and cattle in Denmark. *Nature* 176:156-158. 1955.

11. ———— and ————. Personal communication. 1957.

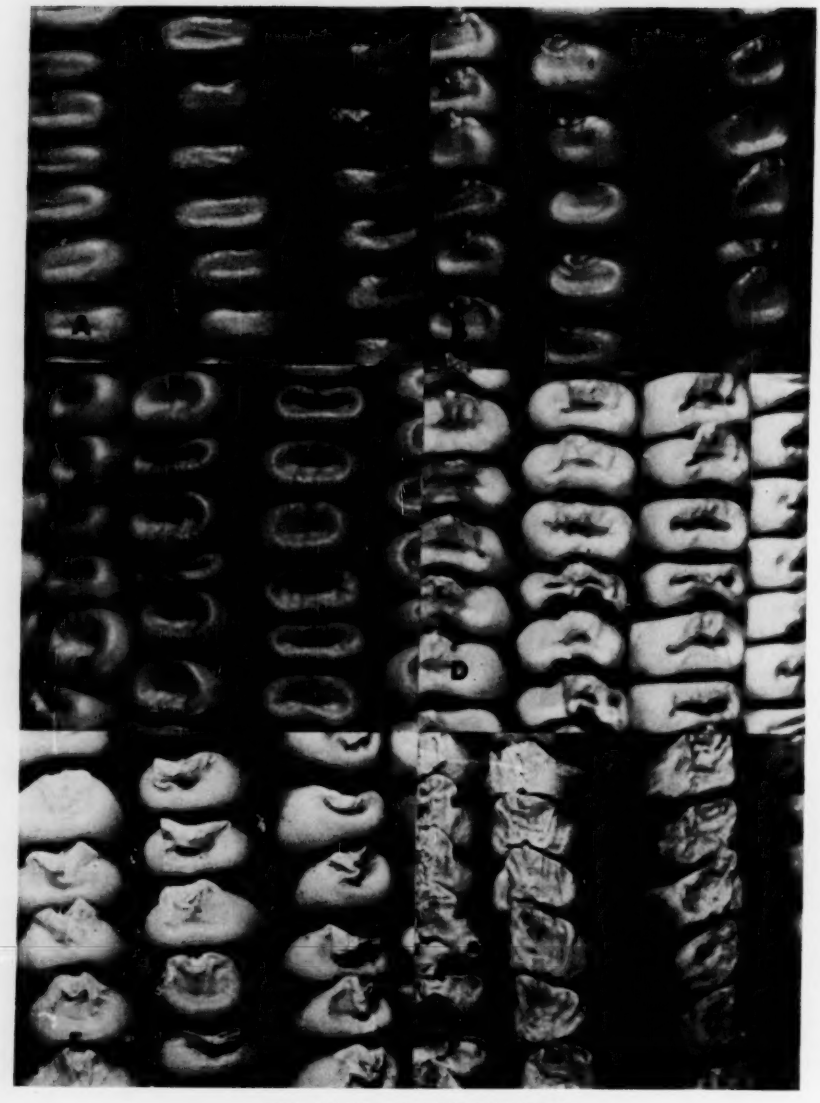
12. Melcer, I., I. M. Orten, R. D. Barner, D. J. Ellis and D. E. Madden. Congenital porphyria in cattle in Michigan. *Science* 1958. (In press).

13. Mitchell, G. E. Hyperplasia of bone marrow and osteohematochromatosis in a yearling steer. *Jour. Amer. Vet. Med. Assoc.* 96: 741. 1940.

14. Plummer, P. J. G. Three cases of osteohaemochromatosis in cattle. *Canad. Jour. Comp. Med.* 13:64-65. 1949.

15. Rhode, E. A. and C. E. Cornelius. Congenital porphyria (pink tooth) in Holstein-Friesian calves in California. *Jour. Amer. Vet. Med. Assoc.* 132:112-116. 1958.

16. Ross, D. B. A suspected case of congenital porphyria (pink tooth) in a heifer. *Vet. Record* 69:345-346. 1957.



PERICARP GRADES IN CORN

Figure 13

Small portions of ears of corn are shown at approximately twice natural size. *A*—grade 1; *B*—grade 2; *C*—grade 3; *D*—grade 4; *E*—grade 5; *F*—grade 6. (Photographs by Mr. Gordon Futral).

INHERITANCE OF LOOSE PERICARP IN CORN

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THE term denting, as it is used in corn literature, denotes indentation of the endosperm as well as wrinkling and sometimes collapsing of the pericarp at the crown of the kernel[1]. It is commonly assumed that shallow denting results in relatively smooth ears, and that the deeper the dent the rougher the ears become.

The results presented in this paper may be helpful in clarifying our understanding of denting, as in this study the roughness of ears is caused primarily by the looseness of the pericarp. This condition has been found to be hereditary.

Methods

The condition of loose pericarp is variable, and to facilitate this study ears were classified according to the following six grades (Figure 13).

Grade 1. The crowns of kernels are smooth, the silk scars are visible but are inconspicuous and the ears are smooth to the touch.

Grade 2. The silk scars are swollen or slightly elongated, and the ears are rough to the touch.

Grade 3. The pericarps are elongated at the silk scar points, and become loose and wrinkled at the tip of the crowns. The ears are rough to the touch.

Grade 4. The elongation and wrinkling of pericarp is much more pronounced than in grade 3. The extra space at the crown of the kernels is filled with endosperm, which gives an ear a very rough appearance, and is very rough to the touch.

Grade 5. The tip of the elongated pericarp becomes somewhat loose, as it is free from endosperm beneath, and the ears are not as rough as in grade 4.

Grade 6. The elongated tip of the pericarp is entirely free from the underlying endosperm, and is usually flattened by the pressure of husks.

The variation in the development of loose pericarp is continuous. The most typical cases are described, but there is a great deal of variation within each group, and sometimes it is difficult to classify the borderline cases. Denting was evident in most of the ears which were examined. It was not intended to assay the relative amounts of the hard and the soft starches of the endosperm; however, a number of smooth ears with predominantly soft endosperm, and nearly flinty ears with loose pericarp were observed.

Experimental Results

Interest in loose pericarp was aroused in 1953 in connection with a single cross test which was conducted at that time. The test involved single crosses among ten yellow inbred lines, including Mpl and GE54.

Inbred Mpl has loose pericarp similar to that of grade 6. However, it is not entirely uniform, and several ears were observed which resembled that of grade 5. Study on the development of the kernel of this inbred indicated that there was no evidence of looseness of the pericarp four days after pollination, while in a number of ears examined from eight days after pollination to maturity loose pericarp was very evident. The endosperm in these specimens occupied about three-quarters of the space in the kernel, while the remaining one-quarter at the tip was free from endosperm, making the pericarp loose at the tip. The layer beneath the pericarp is probably aleurone or nucellus. (Figure 14).

The GE54 ears have prominent silk scars and closely resemble the ears of grade 2; out of 341 ears examined, 9 were of grade 1, 310 of grade 2, and 22 of grade 3.

The GE54 and Mpl inbred lines, and progenies from various crosses involving these two lines, have been grown at Experiment, Georgia each year since 1954. However, to minimize

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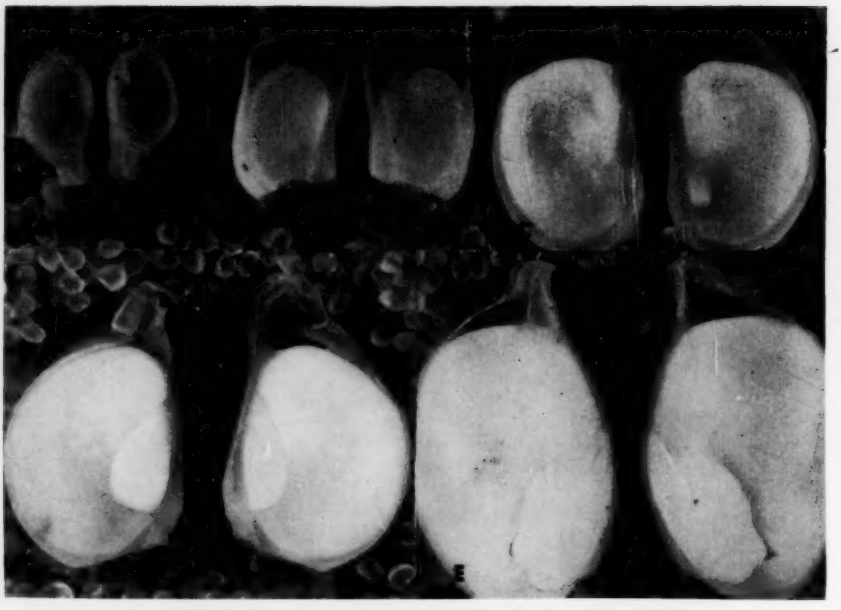

MPI KERNELS AT VARIOUS STAGES OF DEVELOPMENT

Figure 14

Longitudinal halves of kernels at various stages of development, approximately 10 times natural size. Fine sand is in the background. *A*—pistil four days after pollination; *B*—kernel about nine days after pollination; *C*—kernel about 15 days; *D*—kernel about 21 days; *E*—kernel about 30 days after pollination. (Photographs by Dr. Luther Farrar).

the effect of environment, and error in classification, only 1956 and 1957 data are reported here.

The F_1 ears from GE54-Mpl cross had a mean pericarp grade of 3.93, a value intermediate between those of the two parents. The mid-parent value was 3.97 (Table I, rows 1-4). The study of over 40 F_1 ears of various stages of development indicated that in the majority of the ears there was some evidence of looseness of the pericarp, varying from slight to well pronounced, but not as evident as in Mpl.

The F_2 results from the GE54-Mpl cross are given in Table I, row 5. They show a skewed frequency distribution toward smoother grades of pericarp, with the mean grade of 3.70. Both parental types of the pericarp were recovered in a population of 864 ears.

The results of the backcrosses are given in Table I, rows 6-7. The mean pericarp grade of the (GE54 × Mpl) × GE54 progeny was 3.08, which was deviation of .85 of a grade

TABLE I. Frequency distributions for pericarp grades of GE54, Mpl inbred lines, and F_1, F_2 and backcrosses of the GE54-Mpl cross

Row	Inbred or cross	Progeny pericarp grades						Total	Mean
		1	2	3	4	5	6		
1	GE54	9	310	22				341	2.04
2	Mpl					15	153	168	5.91
3	(GE54 × Mpl) F_1		6	87	666	19		778	3.92
4	(Mpl × GE54) F_1		2	28	374	6		410	3.94
5	(GE54 × Mpl) F_2	6	74	192	501	82	9	864	3.70
6	(GE54 × Mpl) × GE54	1	151	239	172	12	2	577	3.08
7	(GE54 × Mpl) × Mpl	1	1	31	379	238	63	713	4.46

from the F_1 mean. The mean pericarp grade of the "(GE54 × Mpl) × Mpl progeny was 4.46, which was deviation of a .53 of a grade from the F_1 mean. These results seemed to agree with the F_2 results, indicating that genes acting in the direction of smoother grades of the pericarp tend to prevail. However, the average of the means of the two backcross progenies was 3.77, which is close to the 3.70 F_2 mean. It was pointed out earlier that the F_1 and the mid-parent pericarp means were quite similar. This indicates the absence of epistasis.

The supposition of stronger gene action in the direction of smooth grades of pericarp is supported also by the preliminary results from crosses of the Mpl inbred with smooth eared corn (Table II). The mean pericarp grade was 1.30 for the F_1 progeny of the Mpl-flint variety cross. From crosses of Mpl with two inbred lines (GE12 and T204) which have smooth pericarp, the F_2 progenies had mean pericarp grades of 2.26 and 2.34. These F_1 and F_2 means are considerably lower than the corresponding means from GE54-Mpl crosses.

There were 25 F_3 progenies from GE54-Mpl crosses grown in 1957, and the results are given in Table III. The progenies are listed in order of the pericarp grade means, from lower to higher values, and are grouped in accordance with the pericarp grades of the F_2 parent ears. These records indicate that significantly different progenies were obtained in the F_3, and secondly, that the pericarp grades of the F_3 progenies in several cases did not correspond to that of the F_2 parents. A situation similar to this was reported for kernel row number in corn. It was shown that the row number of the F_2 parents did not give certain evidence as to the mode of the corresponding F_3 progenies[2]. These discrepancies may be due to the fact that in quantitative characters especially, different genotypes may be phenotypically alike. In the case of loose pericarp, error of classification also is a possibility.

Discussion

Both GE54 and Mpl inbred lines have shallow dented grain, but progenies from crosses between these two lines have a high proportion of rough ears. This seemed to indicate that roughness of ears in these cases was caused primarily by the interaction of loose pericarp with dented endosperm. Our present knowledge of denting in corn and the factors associated with it is still inadequate. Cutler[3] has stated that, "Most difficult to interpret is the occurrence of denting, and until some information on cause and control of denting is available it is impossible to discuss the distribution intelligently." The results presented in this paper indicate that roughness in ears may be experimentally controlled and thus effectively studied as one aspect of the general problem of kernel morphogenesis.

Rough ears are undesirable as they are easily injured during mechanical seed processing. Damaged pericarp promotes deterioration and rapid loss of viability. Seed with damaged pericarp tend, therefore, to give poor stands of plants, and the seedlings are prone to disease. Knowledge of the mode of inheritance of rough ears will facilitate the development of smoother corn.

Summary

The loose pericarp condition in corn is described. Six grades or classes are recognized.

Loose pericarp is inherited as a quantitative

TABLE III. Frequency distributions for pericarp grades in F_2 of GE54-Mpl cross

Parent pericarp	Progeny pericarp grades						Total	Mean
Grade	1	2	3	4	5	6		
2	..	37	..	..	..	..	37	2.00
2	..	13	35	3	4	..	55	2.96
2	2	13	15	14	..	..	44	2.98
3	..	15	19	12	..	..	46	2.94
3	..	15	14	23	5	..	57	3.03
3	4	..	7	14	1	..	26	3.31
3	..	..	91	8	18	..	117	3.38
3	3	7	39	26	29	2	106	3.73
3	..	4	10	39	18	2	73	4.05
3	..	..	2	7	15	5	29	4.79
4	..	8	19	25	4	..	56	3.47
4	..	5	11	14	7	5	42	3.90
4	..	..	2	..	15	15	32	5.34
4	..	..	..	2	11	11	24	5.38
4	..	..	..	5	14	32	51	5.53
4	..	..	..	3	10	22	35	5.54
5	..	3	6	13	28	3	53	4.41
5	..	..	..	3	6	20	29	4.59
5	..	..	1	24	13	14	52	4.77
5	..	..	..	5	22	12	39	5.18
5	..	..	..	3	29	25	57	5.39
5	..	..	2	..	11	38	51	5.67
6	..	..	3	..	19	25	47	5.40
6	..	..	..	..	2	37	39	5.95
6	..	..	..	..	..	52	52	6.00

TABLE II. Frequency distributions for pericarp grades of Mpl - smooth eared corn cross

Row	Cross	Progeny pericarp grades						Total	Mean
		1	2	3	4	5	6		
1	(Mpl × Flint var) F_1	71	11	10	..	..	..	92	1.30
2	(GE12 × Mpl) F_2	2	27	6	3	..	..	38	2.26
3	(T204 × Mpl) F_2	4	46	27	2	..	..	81	2.34

character in Mpl-GE54 crosses. The F_1 progeny has a mean pericarp grade intermediate between that of the parents. The F_2 mean is about equal to the average of the two respective backcross progenies. This indicates the absence of epistasis among genes for loose pericarp, but there is indication of the preponderance of genes for smooth grades of pericarp. Significantly different progenies were obtained in F_3.

The bearing of those results on our understanding of denting is briefly discussed.

Literature Cited

1. Brown, W. L. and Edgar Anderson. Southern dent corns. *Annals of the Missouri Botanical Garden* 35:255-268. 1948.

2. Emerson, R. A. and H. H. Smith. Inheritance of number of kernel rows in maize. *Cornell Univ. Agri. Exp. Sta. Memoir* 296: 1-30. 1950.

3. Cutler, H. C. Races of maize in South America. *Harvard University Botanical Leaflets* 12:257-289. 1946.

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